Selected Essays
on
Orthopaedic Surgery

Newton Melman Shaffer

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Newton M. Shaffer.

SELECTED ESSAYS
ON
ORTHOPAEDIC SURGERY

FROM THE WRITINGS OF

NEWTON MELMAN SHAFFER, M.D.

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With Forewords by Dr. Lovett and Dr. Fisher, and Comments by
Dr. Cotton, Dr. Nutt, Mr. Blagden, and Mr. Hardon

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To The Memory

OF

THEODORE ROOSEVELT, SENIOR

AND

HOWARD POTTER

WHOSE UNOSTENTATIOUS BENEVOLENCE AND PUBLIC-SPIRITED PHILANTHROPY
ENDEARED THEM TO MANY, IN THEIR DAY AND GENERATION, AND
WHOSE KINDLY ASSISTANCE AND ADVICE TO THE AUTHOR, IN THE
DAYS WHICH MARKED THE EARLY STRUGGLES OF THE NEW
YORK ORTHOPÆDIC DISPENSARY AND HOSPITAL INSURED
THE SUCCESS OF THIS GREAT WORK

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FOREWORDS BY

Robert W. Lovett, M.D. Sc.D.
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FOREWORD

By ROBERT W. LOVETT, M.D., Sc.D., Boston, Massachusetts

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The pleasant privilege of writing a short foreword in connection with the part of Dr. Shaffer's work dealing with affections of the bones and joints was offered to me by him and gladly accepted. I had been for a short time, many years ago, assistant surgeon at the New York Orthopædic Dispensary and Hospital, and during that time I had the opportunity of hearing his lectures and seeing not only much of his hospital work, but a good deal of his private work as well. From this experience I learned so much that was of the greatest use to me in my own subsequent practice and teaching that I welcomed the opportunity to write something of those matters which seemed to me in this connection to be most valuable and helpful, and which today I feel are as striking and as true as when they were written.

In the collected writings presented in this volume, tuberculosis of the joints is dealt with in several articles, the first having been published as early as 1877. At this time, it must be remembered, the tubercle bacillus had not been discovered, so that the real nature of the disease was not clearly known, and the terms "scrofulous" and "strumous" were much in vogue. The traumatic element in causation was much advocated, and a clear pathological conception of tuberculous joint disease did not exist. At that time, and more especially a few years later, an operative wave swept over the surgical world, and it was advised by at least one extremist[1] that tuberculosis of the hip should be regarded "in the light of a malignant growth" and dealt with accordingly. As an outcome of this, excisions often followed by distressing results were abundant, and cold abscesses were treated by the most radical incisions.

In this state of surgical opinion, Shaffer calmly and persistently advocated conservative mechanical treatment with a view to curing the disease and conserving function. He pointed out that many abscesses, perhaps most, would absorb under efficient mechanical treatment; that incision was often followed by sepsis and increased activity of the local process; and that the spontaneous rupture of a cold abscess was preceded by a protective walling off of the abscess, prior to rupture. It must be a satisfaction to him to have seen the modern surgical world swing around to a view which, with regard to operative procedures upon tuberculous joints, is as conservative as has been his own.

His description of tuberculosis of the hip is today as clear and modern as it was when it was written in 1877, and the same may be said of his two lectures on ankle joint disease, published in 1882. In the treatment of Pott's disease he held a similarly conservative point of view, and in a monograph on the subject, published in 1879, he advocated the treatment of the disease by the Taylor back brace. It must be borne in mind that in the use of this, and all other apparatus that he employed, he showed a rare knowledge of mechanical principles, and a great accuracy and skill in application.

[1] Barker, *British Medical Journal*, June 9, 1888.

In another field, a most important and lasting contribution was made to our knowledge of static affections of the foot. In 1885 Shaffer described an affection of the foot hitherto apparently undescribed. This was designated by him as "non-deforming club foot." The name was not a very fortunate one, because the condition really was somewhat deforming, and because few people cared to be accredited with any form of club foot. The article never attracted the attention that it deserved, as it was apparently a little ahead of its time; but of late there has been much attention paid to the condition under the names of "contracted foot," "clawed foot," "short tendo Achillis," etc., and all of these articles must begin with a reference to Shaffer's original article. In this article he pointed out (38 years ago) that the affection was often the result of slight, perhaps unnoticed, degrees of poliomyelitis, and today this is being remembered and investigated.

The bearing of a shortening of the tendo Achillis on the occurrence of so-called flatfoot is discussed by Shaffer in an article on that subject, published in 1897; an article years ahead of its time, and one the truth of which was emphasized by our experience in disabilities of the foot which occurred during the war.

He called attention to that important fact that if the dorsal flexion of the foot is checked at the end of the step by an inability of the os calcis to rotate properly, an abnormal and detrimental strain is brought upon the arch, and in all cases of static disturbance of the feet the condition of the tendo Achillis must be taken into account. When this fact, as elaborated by Shaffer, is fully understood by the profession, a distinct stride forward will have been made in our treatment of these affections.

In another field, that of traumatic surgery, there are included in this volume papers of much importance. The disability resulting from elongation of the ligamentum patellæ is described and the symptoms formulated. At the time of the publication of Shaffer's report on this subject the condition apparently had not been recognized. As described by him, it was likely to result from attempts at forcible flexion, under anæsthesia, of partly ankylosed knees. That this state of affairs is of great importance has been brought to general attention by cases in the late war, where attempts of this sort made on knees with adherent patellæ have often resulted in serious damage.

In the paper on the cause and treatment of subluxation of the semilunar cartilages of the knee there is described an apparatus which, in cases for one reason or another are inoperable, is of the highest value in preventing and in many cases curing the slipping of the cartilage. It depends for its efficiency upon checking full extension of the knee, and controlling lateral instability and rotary movements. This apparatus has never been sufficiently known or used on the scale which its merits deserve.

In 1885 Shaffer attempted, by means of a Taylor traction splint, the treatment of an ununited fracture of the neck of the femur of twelve weeks' duration, in a man of forty-two. The patient was kept in bed for the first four weeks, and was then allowed to go about on crutches, but not to sit down. In about three months bony union had occurred, and in six months from the time the splint was applied the patient was discharged as cured, with three-quarters of an inch shortening, but no disability. By those who are familiar with the present and past literature of ununited fracture of the hip and its treatment, it will be seen that this method deserves careful consideration.

The points that have been brought out in what has just been written are simply the ones in Shaffer's work which appeal to me most, and of which I have made most frequent use. Another man would doubtless choose other things, and in the following collection of papers on many subjects each reader can select for himself those which appeal most to him, remembering that they are the contributions of a man of enormous experience, great skill, and one of the eminent medical men of his generation.

FOREWORD

By EDWARD D. FISHER, M.D., New York

Professor of Nervous and Mental Diseases, University and Bellevue Hospital Medical College, New York

In looking over the series of articles of Dr. Shaffer during his active work in his special department of medicine, one is struck by the persistent attempt to get at the etiological factors and thus to diagnose the disease at its earliest inception.

The importance of this is seen in the early treatment thus made possible.

It is interesting to the neurologist to observe his investigations of the involvement of the nervous system in the various forms of paralysis and their consequent joint deformities, as well as in primary joint and bone affections. I can only refer to a few of the many subjects taken up by the author.

Much study has been given to scoliosis. The author has made a classification of the various forms.

He holds that in poliomyelitis there is paralysis and atrophy of the muscles of the spinal vertebræ, due to the involvement of the nerve cells of the anterior horns of the spinal cord. This was an early if not the earliest explanation of the cause of lateral curvature in poliomyelitis and deserves especial mention as this view is now universally accepted. This differs, as he says, essentially from the lateral curvature present in the postural form, which is mechanical in its origin and involves the extrinsic muscles only. The spinal cord type is as incurable as the paralysis in other parts of the body as the upper and lower extremities for example.

The second form usually yields to treatment. I agree with the author in all his views on this form of lateral curvature and etiology.

In regard, however, to Dr. Shaffer's view that scoliosis may often be due to cerebral or spinal disease of the motor tracts, I am not so certain that the etiological factor lies in these tracts.

In diseases of the anterior horns of the spinal cord, we have paralysis and wasting until the inflammatory process ceases. It is an active, progressive process. In the cerebral type of paralysis there is no wasting, except that of disuse, which is necessarily slow and chronic. Again, the electrical reactions are entirely different.

Space will not permit further discussion of this subject.

Dr. Shaffer gives the true explanation of the muscular atrophy in joint diseases. It is essentially neuritic and not of cerebral origin. I would in some degree modify this statement by saying that in some instances there may be a later or secondary involvement of the cells of the anterior horns of the spinal cord by the extension of the irritation along the axones to the cell body of the neuron.

This has been seen in injuries to the nerves in multiple neuritis, due to alcohol, and diphtheritic neuritis with paralysis and atrophy.

The only other subject I can refer to is that of muscular reflex spasm. This symptom is of the greatest importance as it makes it possible to diagnose hip joint disease long before any marked disease can be recognized even by the X-ray; that it is reflex following joint irritation there can be no doubt.

The hysterical element in joint disease, especially in joint affections, is very exhaustively gone into. The diagnosis is made by exclusion, the hysterical form not fitting into any of the established types.

As I have said, in my earlier remarks, the orthopædist has invaded the field of the neurologist, adding much to his knowledge of the functional and organic diseases of the nervous system.

PREFACE

The essays and lectures which combine to make this volume are reproduced exactly as they appeared when originally published, including the footnotes, a few corrections of typographical errors alone excepted.

Where it has been deemed best to omit any printed matter the fact and the cause have been duly noted.

Where any new matter is inserted, as a foot note, the author distinctly calls attention to it.

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PART I

The Spine, the Knee, and Reflex Muscular Contraction

ON REFLEX MUSCULAR CONTRACTION AND ATROPHY IN JOINT DISEASE—WITH REMARKS ON MECHANICAL EXTENSION AND A DESCRIPTION OF NEW APPARATUS.[1]

THE indications for applying mechanical extension in the treatment of morbus coxarius in a direction which corresponds with the long axis of the contracted limb were long since demonstrated.[2] The importance of these indications as designating a principle in the mechanical treatment of those forms of arthritis to which extension and counter extension are applicable has not however, been universally recognized. Mechanical extension in joint disease is capable, if properly used, of accomplishing much good. Dr. D. Prince, in a recent paper,[3] says, "It has done more to lessen the severity and shorten the duration of joint disease than all other things put together." I would add to this statement, however;—when the extension is made subservient to the pathological conditions. Dr. Prince also mentions the fact which Davis demonstrates, and to which I wish to call especial attention, viz. that extension should be applied so as to avoid joint pressure. In my efforts to make extension meet this indication, especially in diseases of the hip and knee joints, I have devised the apparatus which is described below.

The primary contraction that occurs in joint disease is reflex. It has its origin in an inflammation of some of the joint structures. Clinical experience proves that whatever relieves joint pressure and arrests motion, will modify the contraction, and that whatever increases joint pressure aggravates the inflammation and intensifies the contraction. The primary indications are therefore to relieve joint pressure and to arrest motion. These can both be accomplished by a properly adjusted extension apparatus. But if the apparatus be improperly adjusted it is apt to do harm. If we apply extension, for instance, to a diseased hip joint where flexion of the thigh exists, in a line which corresponds with the long axis of the trunk, we create a lever, where the fulcrum (insertion of flexors) lies between the power (extension) and the resistance (joint surfaces). It is with the purpose of avoiding joint pressure in this condition that the limb is placed on an inclined plane—the patient being in the recumbent position. The extension is then exerted, so far as the conformation of the hip joint will permit, directly upon the joint, and the contracted muscles yield, as the cause of the contraction is modified. In a simple case of this character no mechanical device aside from the extension apparatus is necessary, except the inclined plane upon which to rest the limb. Gravity is all that is necessary to overcome the resistance, thus proving that a gentle force alone is required to supplement the extension in overcoming the deformity. When, however, the flexion is complicated (as it usually is) by either adduction or abduction of the limb in coxitis, gravity ceases to be an adjuvant force as applied to these complications. The means that have proved the most satisfactory in these conditions, in my experience, are described further on.

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[1] *The Archives of Clinical Surgery*, June, 1877.

[2] Davis's *Conservative Surgery*, pp. 220 and 221.

[3] "Considerations in Relation to Diseases of the Joints," *American Practitioner*, Feb'y 5, 1877.

We may state it as a rule, which is applicable to all diseased joints for the treatment of which mechanical extension is indicated, that we should never use the contracted limb as the long arm of a lever as a means of exerting any considerable force (power) upon the diseased joint surfaces (fulcrum) to overcome the contracted muscles (resistance). On the contrary, in arranging our apparatus we should bear in mind the anatomy of the joint, its pathological condition and the object to be attained. The extent and direction of the force applied should be governed by these considerations and we should be prepared to gently follow up any remission in the contraction, and thus gradually bring the limb into the desired position.

The employment of a considerable degree of force in overcoming the deformity produced by reflex muscular contraction cannot be too strongly deprecated. If gentle means do not answer, it would be better, in my opinion, to perform tenotomy or myotomy, and thus overcome, at least in part, the resistance, or even to ignore the malposition and to aim only at fixation—leaving the deformity for subsequent operative surgery, than to apply undue force to an already diseased joint, merely for the sake of securing a better position for the limb. Happily the cases are exceptional where the deformity cannot be controlled by measures that will not inflict injury, or aggravate the disease.

In the application of these important principles, in the treatment of joint diseases, I have found the existing appliances imperfect. Dr. Taylor's hip splint affords the best means of making extension in hip joint disease. To it, in 1863, Dr. Taylor added an "abduction screw." A description of the splint and screw are not necessary here. They are both familiar to the profession. It need only be said that they do not, either singly or combined, permit the application of the extension, in many conditions that present, in the *exact* line of the deformity. I have devised what I have called a "lateral screw," which allows extension in *any* direction that is indicated, and which at the same time can be made to act, without removing the splint, as an *adductor* or an *abductor* of the limb, at the pleasure of the surgeon. It can be attached to Dr. Taylor's splint in the same manner as his "abduction screw."

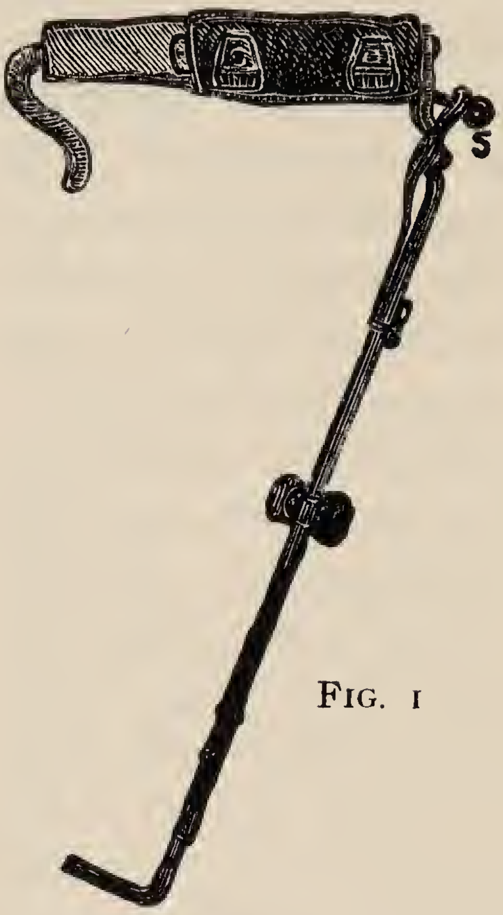

Fig. 1

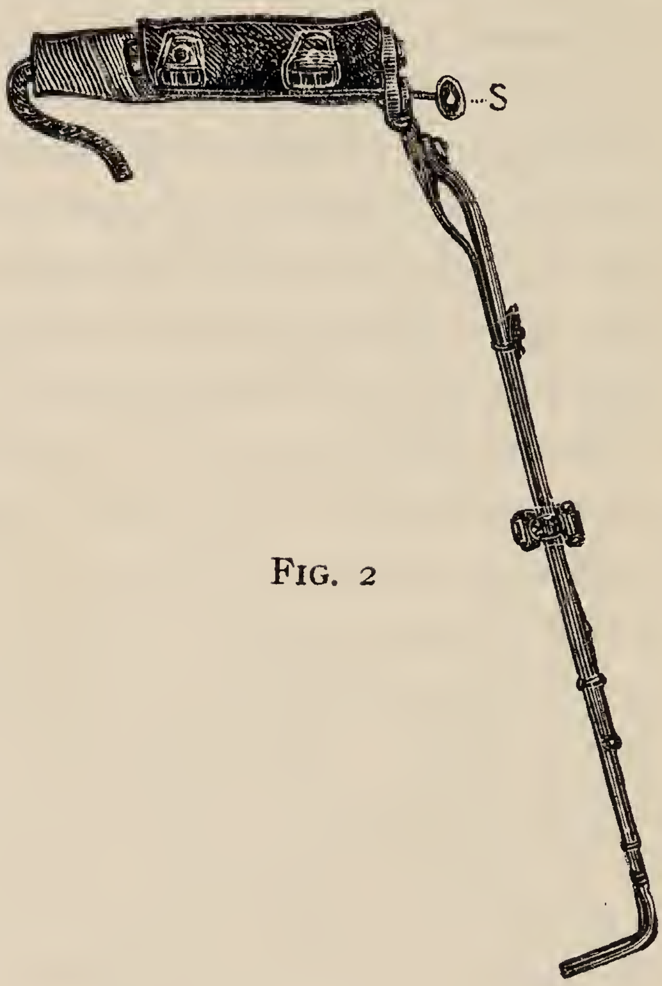

Fig. 2

Figs. 1 and 2 represent the "lateral screw" as applied to one of Taylor's splints. 1 shows the shaft adducted,—2 abducted. By simply turning the screw at S. the shaft of the splint can be made stationary at any point between the extremes indicated in the engravings. By using the screw, when the splint is adjusted, the surgeon is enabled to gradually and very gently change the position of the limb at will.

The "lateral screw" (Fig. 3) consists of two parts, A and B, joined together by the lateral hinge C. The part A is fastened to the pelvic band. The part B is attached to the shaft of the splint. Through the everted lip D, there passes a screw S, which operates through a button (which revolves on a horizontal axis), and which is fastened into another button (also revolving on a horizontal pivot) in the part A. By turning this screw we can either approximate the lip D toward the part A (producing *abduction*), or by reversing the screw we can separate D from A, and *adduct*. E, E, represent the screw bolts, by which the apparatus is attached to the hip band and shaft of the splint.

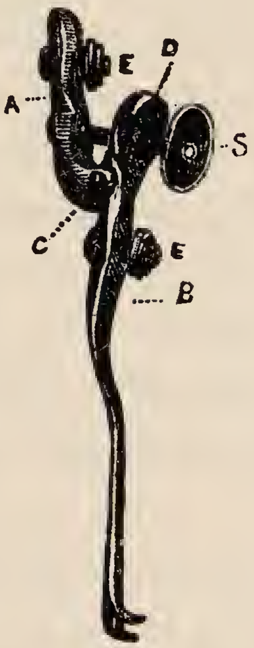

FIG. 3

Before the splint is applied the screw should be turned as the circumstances indicate until the direction of the shaft of the splint corresponds with the position of the limb. After adjustment, extension is applied, in the "line of the deformity," and no attempt should be made to overcome the contraction by the use of the screw, for several days. It should then be used very gently. It is better to imitate the force of gravity, by which flexion is generally so readily overcome, and only "follow up" the contractions as they are modified by the extension, immobilization and the proper therapeutical adjuvants. If we use the screw to *abduct*, the ordinary perineal pads, which form the basis of the counter extension, will also be the point of resistance. When we use the screw to *adduct*, it will be necessary to supplement the *perineal* with *shoulder* straps, and to apply a little more extension than is required, so that as we use the "lateral screw," the extra force may be transferred to and lost upon the shoulders. Adequate extension and adduction are thus obtained. We should also secure the hip band firmly by the padded strap, which completes the circumference of the pelvis. In order to secure the shoulder straps in position, we cross them as they pass upward from the hip band to the shoulders on both the anterior and posterior walls of the thorax. It is also necessary to secure the pelvic band, so that its anterior half will form a slightly acute angle with the shaft. This may be done with webbing straps.

In a case of morbus coxarius (Mary McDonald, aged 8), occurring in my service at St. Luke's Hospital, the limb was abducted so that the external malleolus was removed 17½ inches from the median line. The limb was rigidly held in this position (with slight flexion), and no motion whatever at the joint could be detected. Taylor's splint, with the "lateral screw" was applied, as above described, in the *exact* line of the deformity. I then made exaggerated direct extension, and immediately proceeded to adduct the limb with the screw, thus transferring the extra force, which would otherwise be exerted on the *perineum*, to the *shoulders*. The result was an immediate and painless adduction of the limb to the extent of three inches. On the second day, with the splint removed, the limb assumed under gentle manipulation, unaided by any artificial support, a position six inches nearer the median line. In other words, extension in the direct line of the deformity, with a painless adducting force had, in twenty-four hours, relieved the deformity to this extent. The limb was thus gradually adducted until the deformity was overcome in about ten days. This case was an extreme one, so far as the deformity was concerned, and I selected it as a case in which to test the efficiency of the "lateral screw" as an *adductor*. The degree of the force applied was unnecessary. But as no pain was excited and no constitutional symptoms supervened, we felt justified in making it a case in which to note the result of pressure upon the shoulders, and the necessary arrangement of the entire apparatus. The result was extremely satisfactory. The patient is now walking about in a plain Taylor splint—the screw having been removed. In another case, also in St. Luke's Hospital (Mamie Fitzgerald, aged 4½), I applied the "lateral screw" as an *abductor*. The limb was adducted and flexed to an extreme degree. By a cautious use of the direct extension with the "lateral screw" the limb was easily straightened, with no unfavorable symptom, and without pain.

In all cases, the inclined plane should be used to accommodate the contracted flexors. I generally use for that purpose two or more tolerably hard pillows arranged to meet the indications.

The anatomy of the hip-joint, and the relations to it of the neck and shaft of the femur, render the problem of controlling extension upon this articulation, in the manner indicated, a rather difficult one. As we make extension with Taylor's splint, the force passes to the trochanter major, in the line we would choose. But here the angle of the neck, and the ball and socket articulation at the extremity of it, interfere with the transmission of the force in a direct line. In extreme adduction of the thigh, for instance, where the distal extremity of the limb lies inside of the mesial plane of the body, the inferior portion of the joint would be subjected to a pressure as a result of extension, that would be equivalent to the force applied. The difficulties in the way of making extension in the line of the neck are obvious. For these reasons we should be very guarded in the use of that which we apply in the line of the shaft of the femur. I have seen several cases where a disregard to these simple anatomical facts, has aggravated the disease. Under any circumstance we should not depend on extension wholly. Recognizing as a fact that, in by far the greater number of cases, the initial lesion consists of an inflammation in the cancellous structure of the bone, and regarding traumatism as an exciting cause, I do not depend on mechanical means alone in the treatment of an essentially constitutional disease.

The positions assumed by the thigh in morbus coxarius (as a type of joint disease) are due, I believe, to the reflex contraction. In other words, we have associated with the joint disease and dependant upon it, an inflammation of the peripheral nerves supplying the joint, and that the reflex contraction as well as the direct atrophy are but the expression of the nerve lesion. I may state as a result of my clinical observations on this point, that the force and persistency of the contraction, the muscles affected by it, the degree of muscular atrophy and the rapidity with which it occurs, and the extent of the impaired electro-muscular contractility, all have their value as indicating the actual pathological condition of the diseased articulation, and are of great service in making both our diagnosis and prognosis. In chronic osteitis, especially if associated with a chondritis, the contraction is firm, tense and very persistent. It increases with greater or less rapidity until anchylosis is simulated. The atrophy is slowly but steadily progressive and the muscles show a marked decrease in Faradic reaction. This is especially the case in the dry osteitis. In chronic (gelatinous) synovitis, the contraction is not so marked, and the muscles do not so quickly respond as they do in chronic osteitis, to the stimulus of passive motion. The movements of the joint are more or less free and within a limited area, are not productive of pain. The atrophy also occurs slowly—more slowly than in the chronic epiphyseal inflammation, and the reduction of the Faradic reaction is not so great. The trophic disturbances are in accord with the sluggish nature of the lesion. In acute synovitis, however, the atrophy occurs sometimes with surprising rapidity, and the contractions sympathize with the acute character of the disease. Paget says: "This wasting occurs quickly in nearly all acute diseases of the joints—more slowly in the chronic inflammations."[1] Again, when speaking of acute joint disease: "It is, I repeat, not a mere wasting from disuse—it is far more rapid than that, more like what has been called acute atrophy of muscles, such as is seen in the swiftest cases of infantile paralysis."[2] This wasting, which Paget calls "reflex atrophy," "seems dependant on disordered nervous influence, and often appears proportionate to the coincident pain, as if it were due to the disturbance of some nutritive nervous centre, irritated by the painful state of the sensitive nerve fibre."[3] Coincident inflammation, rather than "coincident pain," would, I believe, have better expressed the condition.

Brown-Sequard was the first to propose and demonstrate the following propositions: "*Nerve irritation alone*

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[1] *Clinical Lectures & Essays*, page 208.

[2] *Op. cit.*, p. 209.

[3] *Op. cit.*, p. 209.

is capable of determining rapid and early atrophy of the muscles, preceded by decrease or disappearance of Faradic contractility. Complete nerve division does not induce atrophy and loss of electrical contractility until after an incomparably greater lapse of time, as in the case of prolonged inaction."[1]

Charcot remarks: "If lesions, whose consequence is the abolition or suspension of the action of the nervous system, are impotent to produce in distant parts other nutritive disturbances than those attributable to prolonged inaction, *it is not thus as regards lesions, which determine either in the nerves or nervous centres, an exaltation of their properties, an irritation or an inflammation.*"[2] Again, he says: "It appears in fact to be demonstrated that the latter [muscular alterations consequent on nerve-irritation] supervene with much greater rapidity, and are preceded and accompanied by more or less marked modifications of electrical contractility, which do not show themselves in the former [muscular alterations connected with functional inertia] with the same characteristics, and only make their appearance at the end of a very long lapse of time."[3]

The atrophy that occurs from simple functional inertia combined with the pressure produced by adhesive plaster and bandaging, is witnessed when the not infrequent error is made of treating by these means and an apparatus, a neuromimesis of a joint lesion, for the real disease. This simple atrophy following disuse and pressure is altogether different from that which ensues from an actual joint lesion, and is unaccompanied by a loss of electro-muscular contractility. These facts I have demonstrated to my own satisfaction in many cases. But in order to secure the opinion of a high authority, I invited my friend, Dr. E. C. Seguin, to test several cases occurring in my service at the Orthopædic Dispensary. Four cases of hip-joint disease [none of which had been tested by myself], selected at random, were examined by Dr. Seguin, in presence of Drs. M. R. Vedder, of N. Y., A. G. Thompson, of Islip, L. I., and the house staff of the hospital. In every case, the muscles of the *thigh* showed a very decided decrease of Faradic contractility, while those of the *leg* showed a normal reaction. Here again was shown the difference between the trophic disturbances following joint disease and those which ensue upon simple disuse. All of these cases had been under treatment for a long time—the affected limb being subjected to the pressure both from adhesive plaster and bandages—and kept in a position where no weight had been thrown on it for many months.

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[1] "Lectures on Diseases of the Nervous System," by J. M. Charcot. New Sydenham Society, 1877, pp. 13 and 42.

[2] *Op. cit.*, p. 12.

[3] *Op. cit.*, pp. 37 and 38.

I have recognized these facts for a long time, and it has been my custom to instruct the house staff at the Orthopædic Dispensary and Hospital, to ignore muscular atrophy, so far as treatment was concerned. Properly applied extension will prevent rather than augment that which is due to the disease. We certainly, in view of the fact that it is unimportant compared with the disease we are treating, can afford to ignore that which occurs from disuse alone. I have under my observation now a case of joint disease in the adult, of over fifteen years' duration, where supports of various kinds, or crutches, have been constantly used. There exists a slightly reduced Faradic reaction only in those muscles which are unconnected with the diseased articulation. As Charcot remarks, the nutritive disorders of the muscles consequent upon functional inertia make their appearance at the end of a *very* long lapse of time.

The value of these facts, as a means of diagnosis and prognosis, I shall make the subject of a future paper.

A case of traumatic hip-joint disease sent me by my friend Dr. Leroy McLean of Troy, N. Y., will illustrate the relation of the reflex contraction and the atrophy to the lesion.

Mr. F. H. P. of Glens Falls, N. Y., a strong and healthy man of 24, fell, on the 4th of November last, through an elevator well, a distance of 36 feet. The force of the fall was expended on the left hip-joint in a direction upward and outward. No dislocation or fracture occurred. After the fall he was assisted to rise and walked several steps. The immediately urgent symptoms following were those of intestinal obstruction, though there was an ecchymotic line about two inches broad, extending from the inside of the thigh to the hypogastric region. No acute joint symptoms were present. After a time there was an uneasy sensation about the joint with pain in the knee, and the limb began gradually to assume an adducted and flexed position. The adductors and flexors became "tight" and the latter "stood out like large cords." At the end of three weeks the patient got up and moved about on two crutches, just touching the toe to the floor. He used crutches for three weeks, and afterwards, a cane. He finally, ten weeks after the accident, discarded all artificial support and walked unaided, with a deformed and apparently shortened limb. But as exercise was thus continued, troublesome symptoms began to appear. Any sudden movement of the limb gave pain, especially at the knee, and during the night he was frequently awakened by it. The limb became more deformed, the wasting increased, and the joint grew more and more rigid. The general health was also affected. At the examination of the patient (April 7th last) I found the joint apparently anchylosed with marked adduction and slight flexion. The most careful tests gave no evidence whatever of motion. The flexors and adductors were rigidly and prominently contracted. As Dr. McLean had only a short time before administered chloroform, and as he informed me that the contraction wholly disappeared under its use (only to return, however, with consciousness) I did not administer an anæsthetic. I should have done so but for this positive information. The Doctor also stated that no roughness of the joint surfaces or crepitus was discoverable. Careful measurement showed that no shortening had occurred. The atrophy of the muscles of the thigh was marked, and (as was subsequently ascertained) their electric contractibility greatly impaired. Faradic reaction was normal in the muscles of the leg. The patient complained of uneasy sensations about the joint and at the knee, and the symptoms mentioned above were all present, aggravated by the natural progress of the disease. Carefully applied extension in the exact line of the deformity (using the "lateral screw" and inclined plane) has corrected the deformity, relieved the pain and re-established a considerable degree of motion. This motion is limited, however, by an involuntary and very sudden contraction, principally of the adductors. The patient (who is very intelligent) likens the sensation to that experienced when one anticipates a blow upon the epigastrium, which, however, is avoided. He has made extraordinary attempts to allow free passive motion of the joint, but the involuntary check invariably occurs at a certain point, not from the pain actually inflicted, but from the apprehension that it will occur. We have here a hip-joint lesion from the differential diagnosis of which synovitis, both acute and chronic, may be eliminated and the cause of which is distinctly traumatic. The symptoms, disregarding entirely the reflex contractions, pointed unmistakably to an osseous lesion of the hip-joint. I can not doubt that it is inflammatory. The reflex contractions (yielding wholly under chloroform) were so firm and tense as to simulate an actual synostosis of the joint. The atrophy has been progressive and exceeds to a considerable extent that which would be induced by simple disuse—and the greatly diminished electro-muscular contractility of the muscles of the thigh (those of the leg remaining normal), together with the history of the case, forms an interesting contribution to the study of joint diseases. We add, in conclusion, that these same conditions of reflex contraction, simulating anchylosis, slowly progressive atrophy and impaired Faradic contractility, exist also, in a marked degree, in those unmistakable cases of dry osteitis which affect the hip-joints of children.

The remarks occurring in the first paragraph of this paper are peculiarly pertinent to those lesions of the knee-joint for the treatment of which extension and counter extension are applicable. The various appliances which have been used in treating the diseases of this articulation are, as a class, very faulty in principle. Here again extension is generally applied in such a way that the joint surfaces are made to bear the pressure used to overcome the contraction—the line of extension as ordinarily applied forming one side of a triangle, the other two sides of which are parts of the thigh and leg, respectively. The resistance to be overcome lies in the flexors—notably the biceps. Again, we see a lever established whose fulcrum is at the joint surfaces, the use of which in extension produces joint pressure and directly favors sub-luxation of the head of the tibia backwards.

I have attempted to meet the pathological indications presented in knee-joint lesions with an apparatus which I devised sometime ago, and which has answered so excellent a purpose in many cases that I feel warranted in calling the attention of the profession thereto. Its design is to apply the desired force directly to the head of the tibia,

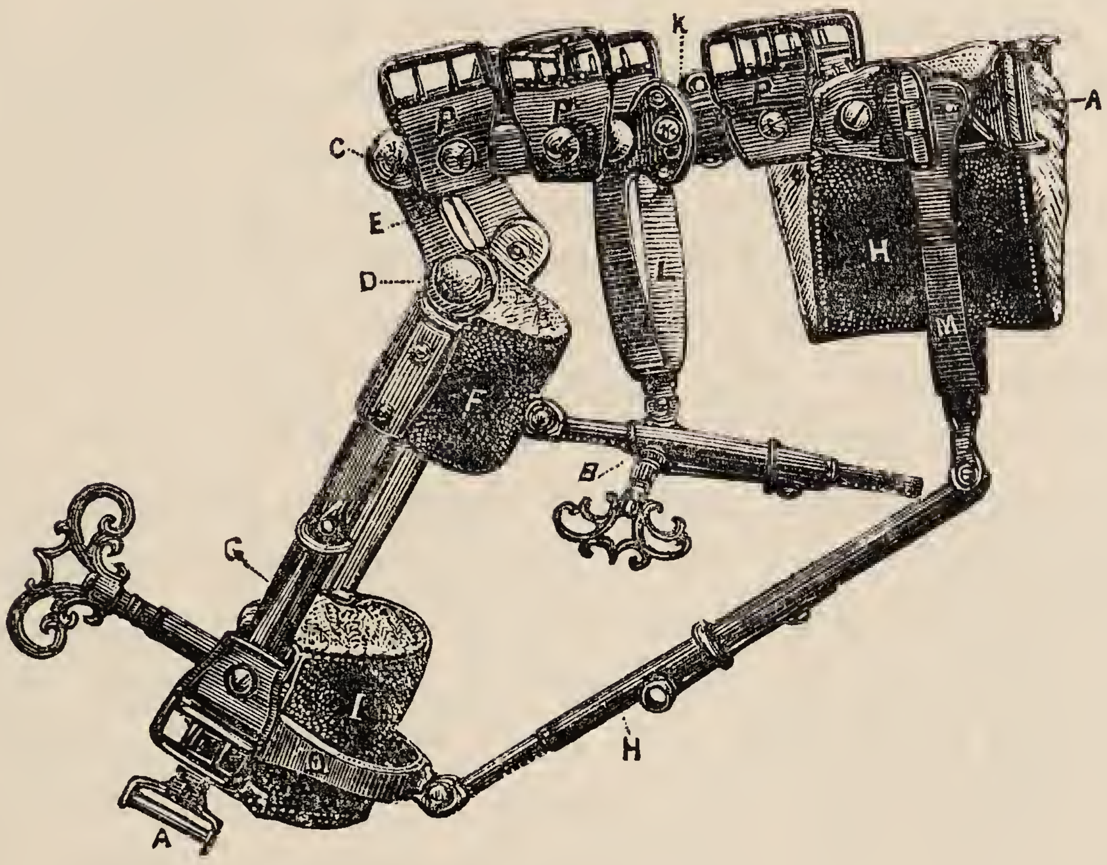

FIG. 4

throwing the same forward and downward by a simple movement. While we thus overcome the muscular contraction *in a direct line*, we relieve the joint pressure and overcome the deformity simultaneously. The apparatus is represented in figure 4. It consists of three principal parts; the thigh, leg and intermediate. The first two are secured to the limb by adhesive plasters which are attached at the points A, A. Extension is made with a key at the extension rod proper at B. The joints at C and D move upon pivots, and as the extremities of the apparatus are secured by their adhesive straps at A, A, the joint D moves forward and downward, describing the arc of a circle, the radius of which is the bar E. Pressure is thus made directly upon the head of the tibia by the band F, and this can be very greatly augmented by using the extension rod at G, which further relieves the joint of pressure by additional extension in the position already acquired by the preliminary extension of the rod B. H is an accommodation—not properly an extension rod—which glides forwards as the extension is applied at B. As soon as the leg is thrown sufficiently forward, the accommodation rod is secured by a slide, and an extra turn of the key at B and G leaves the joint free from pressure and with adequate extension applied directly to the contracted flexors. The thigh and leg bands at H and I move upon pivots so that they adjust themselves readily to any position, and at K there is an arrangement by which the curved bar L may be adjusted to suit the requirements of the extension rod B. The bars M and O are secured to the thigh and leg parts by double rivets. Through the buckles at P, P, P, webbing straps (padded) are passed, producing counter extension in addition to that secured by means of the adhesive straps. The Faradic current applied to the muscles in diseases of the knee-joint, shows (so far as my experience goes) a decrease in contractility of the gastrocnemius, biceps, semi-tendinosus and membranosus, and also a slower reaction in the quadriceps.

A LECTURE ON LATERAL CURVATURE OF THE SPINE[1]

Delivered at the New York Orthopædic Dispensary and Hospital, December 17, 1880

ETIOLOGY AND PATHOLOGY

There is no condition among those recognized as orthopædic that demands more careful consideration than lateral curvature of the spine—there is none, the etiology of which is so obscure or the pathology of which is so much in doubt—and finally, though no other condition known to chronic surgery presents so little danger as to life, there is no other the treatment of which is so unsatisfactory. It is a veritable *pons asinorum*, and orthopædic surgery cannot occupy its proper status as a scientific branch of surgery until the bridge is passed.

I approach the subject, therefore, with some hesitation, for I cannot, to-day, answer all the questions that should be answered. There seems to be so little to the superficial observer to be accounted for, while there are, in reality, deeper problems than have yet been sounded. We shall, at least, look at the conditions practically, and when we are in doubt, we shall say so. In no other way can we expect to reach satisfactory conclusions, for, even if we cannot explain all the phenomena of lateral curvature to-day, we can, at least, place on record certain observations which may aid others, and which will also be of value to ourselves in the future.

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[1] *The Medical Gazette*, April 2, 1881.

At the very outset we are placed at a disadvantage in an anatomical sense. Our effort to find any one who has thoroughly mastered the muscles of the back is like the traditional search of Diogenes. In studying the anatomy of almost any other region we may rely upon the recorded observations of thorough students which have been verified time and again, but works on anatomy are conspicuously deficient as regards the regional mechanism of the spine as influenced by muscular action, though much valuable information has been contributed as to its general mechanism. We must, therefore, begin at the very beginning.

In doing so we must not err, as others have done, and view the spinal column stripped of its muscular appendages, but consider it as it is in life—we must, further, study the difference that exists between functionally weakened muscles and structurally changed muscles and we must appreciate the effect produced in other parts of the body by these marked different states. We must know what produces functional muscular weakness and structurally shortened muscles, and with this knowledge gained by study in other parts of the body, we must apply our deductions to the conditions found in lateral curvature. In this way we shall be liable to no gross errors. We must reach conclusions, in the absence of reliable post-mortem descriptions, by analytical study of clinical facts and the application to them of physiological and pathological laws.

Nothing is absolutely known or definitely settled regarding the etiology of lateral curvature. Certain facts are known which, in themselves, have been interpreted as having an etiological value but they do not stand the test when applied to the conditions which they accompany. It is known, for instance, that lateral curvature develops most frequently in females between the ages of 12 and 18 years, that it presents as a deformity involving the right side more frequently than the left, and that a peculiar nervous state, called hysterical or emotional, almost always accompanies its development. But the relation of age to this particular lesion is not at all peculiar; almost all chronic diseases have a similar history in this regard; poliomyelitis is rarely anything else than a disease of infancy; joint and spinal diseases are typical lesions of childhood, and lateral curvature develops much more frequently in children and in boys than is generally supposed—with sufficient frequency, anyway, to warrant us in saying that its etiology has nothing to do with either age or sex. It is fair, however, to state that the conditions that finally produce lateral curvature are more likely to exist in girls who are just approaching or who have just passed puberty, but there are many conditions present, just at this particular time, which may have no direct relation either to the sexual development, or to the strictly physiological processes then occurring. If typical lateral curvature can exist and progress in babies of 6 months, or in boys of 8 years, or in young men of 18, we must take a much broader etiological view than has heretofore been held. So also the greater frequency of the curve to the right; it occurs, not infrequently, to the left, and that without any cause that the most searching physical examination can detect; they are not "left handed" cases; there is a normal relation of the thoracic viscera, and there is no evidence, in any case I have ever examined which enables me to determine why the curvature presents more frequently to the right than to the left side. Holden remarks that there is a normal curve of the spine to the right at the 3d, 4th, and 5th dorsal, the reason for which is not clear; says Holden: "The solution of the question is of no practical value—all we need remember is that the curve is natural." This fact should be borne in mind and its value duly estimated in our study of the etiology of lateral curvature. There is, probably, some relation between this physiological curve and the pathological condition; what, we do not know, but we may say, at least, that this curve is not an important factor in the production of the curvature found. At most, it has a co-incidental relation and may be one of the determining factors in the direction of the curve. Moreover, this curve is strictly lateral and does not present the elements of rotation, or of other conditions found in lateral curvature, and is unaccompanied by any loss of lateral flexibility. It is a simple, physiological curve, and is present, I believe, in many instances.

There is the third important and constantly present condition found in lateral curvature, viz.: the hysterical or emotional state. By many this has been supposed to be the cause of the curvature. The methods by which this condition has been explained as the potent factor are as various as the writers who have adopted it, but we may dismiss it from the catalogue of efficient causes. It has the same value in lateral curvature as mental precocity in the strumous diseases of children. It points to the general condition, and is an index of that condition, or of a condition of the nervous system. All children that are precocious, as to intellect, are not strumous, nor do all hysterical or emotional girls develop lateral curvature. We must look further and deeper than this to find a cause that will explain the conditions found.

Abnormal positions in school, at the piano, etc., have been credited with causing lateral curvature, but here, evidently, effect has been mistaken for cause. Position, unless long maintained without change, does not result in permanent deformity. However bad, in many respects, the position at the school desk may be, the school girl spends many hours out of the twenty-four away from it; hence, if a bad position is maintained, there is some other cause than simply mal-position at work. If a young girl habitually occupies a bad position when standing (and there is no difference in the length of her limbs) the cause or causes of lateral curvature, whatever they may be, may be in active operation, and, unless controlled, a true curvature may result. But while simple mal-position is frequently the first sign which many patients give of the progressive lesion that may have existed for a long time and which results in true lateral curvature, there are many instances where this peculiar attitude is due to simple functional weakness of the spinal muscles, which is as unimportant as is functional weakness in any other part of the body.

The fact also, that the acquired position, produced by an unequal length of the lower extremities, does not result in true lateral curvature, is incontrovertible evidence that nothing need be feared from mal-position alone. In cases where arrested development from infantile paralysis, hip-joint disease, etc., produces a shortened limb, there is only a symptomatic lumbo-dorsal curve, which disappears when the patient lies down. I have never known a case of permanent lateral curvature to develop from this cause. I have measured, in more than one hundred patients, the relative length of the lower extremities in cases of the true disease, and found no greater difference than exists in what is known as the normal condition; *i.e.*, slight differences, in no case more than three-eighths of an inch; and in only two cases was that extent reached—and there were instances where the difference was *curative*, that is, the shortening was on that side which antagonized the true lateral (dorsal) curvature above. Each of these cases came to me with a cork sole to the shortened limb, for the difference had been recognized but the effect had not been studied. Each had a dorsal curve to the right and the shortened leg was on the same side. Of course, if the difference in the length of the extremities were a factor, either the shortening would have been on the left or the curve would.

The fact, therefore, that true lateral curvature is not associated with unequal length of the lower extremities while those conditions that produce the inequality are unassociated with it, proves, conclusively, that simple mal-position is not a factor in the production of scoliosis—and it furthur demonstrates that unequal muscular action of the long, extrinsic muscles of the spine, unassociated with a cause that affects the vitality of those muscles, does not produce it. So long as there is functional trouble only and no disturbance that produces either a state of tonic contraction or of paralysis, there need be no fear of true lateral curvature.

Reviewing, then, the whole field, we find that none of the facts or conditions we have discussed can account for true lateral curvature:—the age is very variable; both sexes are its victims; the deformity is not confined to either side; the emotional or hysteric state is too general to be of any value and mal-position cannot be considered as being of any etiological importance. To what then shall we look for an explanation of the phenomena of the disease?—for it is what we can see rather than what the patient feels, that is to be of use to us in reaching even approximately correct conclusions.

What can we see in the condition known as true lateral curvature? We will state the conditions and, from them, attempt to draw conclusions:—I. There is a curvature of the spine either to the right or left, involving, generally, the portion between the scapulæ. This, for reasons to be stated further on, we regard as the real curvature, the others being merely secondary.

II. There is a rotation of these vertebræ upon each other, the rotation being always towards the convexity of the curve. This can be demonstrated by examining the transverse processes.

III. There is more or less mal-position of the scapulæ, the one on the side of the convexity being thrown further away from the spinous processes, the other being nearer them. There is also an undue prominence of the lower angle of the scapula on the convex side of the curve, which is frequently the first sign observed. The cause of these altered relations of the scapulæ to the spine can easily be traced to the altered relations of the ribs, which are placed in an abnormal position by the rotation of the vertebræ, those on the concave side being depressed, while the others are rendered more prominent.

IV. Actual atrophy of the mammary gland on the side of the prominent scapula, and always on the side toward which the convexity looks and toward which the bodies of the vertebræ are rotated. This I have observed and demonstrated in many cases. It is a sign that is very rarely wanting when the primary curve is in the dorsal region.[1]

V. This lateral dorsal curve is a rigid curve, associated, apparently, with disturbed intrinsic muscular action, as can be demonstrated by various tests, such as forced lateral flexion in a direction that tends to straighten the spine, or by suspension. In many cases this rotated and laterally curved spine cannot be straightened by any means we dare employ, though we can modify it, and one of the efficient causes which prevents the reduction is this rigid, intrinsic muscular resistance. This muscular resistance is absolute, it is a true contracture, unmodified under ether or chloroform, unlike the reflex spasm of chronic osteitis of the joints. It affects the muscles chiefly of the concave side of the deformity, or, at least, the apparent effect is here.

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[1] I have recently seen cases where the true curve was in the dorso-lumbar region, in which the atrophic changes were easily apparent in the lower extremity on the side toward which the convexity looks:—in other words, *on the paretic side.*

VI. There is always a compensatory curve above and below this rigid dorsal curve, but these curves are compensatory only, as can be proved by testing the degree of mobility at these points. Furthermore, there is little or no true rotation at these places where the compensatory curves exist.

VII. After this condition has existed for a long time, the long muscles of the back show, under ether, a structurally shortened state which does not exist in the earlier stages, for then the lesion, whatever it may be, affects only the region where the true lateral curve exists. There are also other secondary signs to which I could call your attention, but they are so truly secondary and so purely symptomatic that I shall consider them under the head of symptoms.

We have, then, to explain the progressive, rigid, lateral curve, the rotation, the disturbed muscular action and the atrophy of the mammary gland.

Let us now consider what is discovered by post-mortem appearances in advanced lateral curvature, for I have been unable, in all my researches to find any recorded evidences of what is found in the early stages, such as I have been considering. They are simply these:—a wedging together of the articular processes on the concave side of the curve and very marked evidences of muscular degeneration in both the intrinsic and extrinsic muscles. There is no evidence adduced to show disease of the bodies, laminæ or pedicles or of the transverse or spinous processes or of the ligaments. The osseous lesion lies wholly at the articular processes, and the question to be decided, the solution of which would answer many other questions absolutely, is this:—Is the articular lesion primary, or is it secondary to a cause which has produced a progressive contracture of certain extrinsic or intrinsic muscles?

If the osseous lesion were primary, it would at once account for the rigid curve, though it would be difficult to account for its progressive character, the rotation or a disturbed muscular condition, which finds expression in a contracture rather than in a *reflex muscular spasm*, such as is seen in chronic osteitis of the joints, in fractures of the long bones and which yields under ether. Nor would it readily explain the almost uniformly present mammary atrophy and especially on the side opposite to that on which the osseous lesion occurs. For example: the articular processes are wedged together on the concave side of the curvature—generally the left—while it is the right mammary gland, that is atrophied under these circumstances. Were it otherwise, we might attempt to explain the wasting, on the ground that the spinal nerve or nerves were secondarily affected by pressure or by inflammation, but we have here, at once, a symptom which we cannot explain on this hypothesis, for I am positive of this actual atrophy of the mammary gland in true lateral curvature. I have carefully tested it in many cases and find only one or two exceptions, and when the convexity of the curve is to the left, the mammary atrophy is on that side, and, in my experience the rule holds good that the wasting of the mammary gland is on the side of the convexity of the curve. I have not even one exception to record on this point.

If we take the other view, and regard the osseous lesion as secondary to the disturbed muscular conditions, we are not met by so many difficulties.

We will assume, therefore, that the rigid curve is produced by this muscular action, viz.: a unilateral contracture of some of the intrinsic muscles. What is it that usually produces a unilateral contracture? Is it not a paralysis of the antagonists? There must be either some essential loss of power by these antagonists or there must be an exalted state of the shortened muscles. But loss of power—paralysis—is usually accompanied by some trophic symptoms, and we have, I am very sure, in this condition of true lateral curvature, an actual loss of power in certain muscles, and a progressive contracture such as we see in infantile paralysis, torticollis or congenital club foot. Some of these intrinsic spinal muscles are also strong rotators. The same *contracture* that produces the lateral curve also produces the rotation, and, accompanying this loss of power on the convex side, we find not only the paralysis but also the trophic symptoms. These are most evident in the mammary gland, though I doubt not, if sufficient care be used, they will be found at other points also. It is not until lately that these trophic changes and their relation to the lesion have struck me so forcibly. I shall investigate the matter thoroughly and shall tell you the results at some future time.

So that we have a solution of all the important phenomena of lateral curvature upon the basis of disturbed muscular action involving a localized loss of power on one side, and a localized progressive contracture on the other.

The primary rigidity of the curve is easily explained. The subsequent rigidity and anchylosis of the articular processes is the result. The rotation follows as a matter of sequence and occurs early before the anchylosis. The trophic symptoms are on the side where the paralysis exists, and finally the progressive character of the disease, the great difficulty of arresting or controlling its progress and the resulting great deformity can only be explained on the hypothesis we have so imperfectly advanced.

But what produces this unilateral paralysis? why does it affect girls more frequently than boys? Why is the right side more frequently affected than the left? Why should it develop more frequently at a particular time of life? and why should the hysterical and emotional be peculiarly its victims? I cannot answer all these questions. They must await the careful and scientific investigation that alone can solve them. I might spend much time in speculation here, but forbear to tire you. I will, in closing this portion of my lecture, merely call your attention to the very evident error that many writers make in attempting to trace true lateral curvature to the action of the extrinsic muscles of the spine. These muscles, which have their origin at some other part of the skeleton and their insertion at the vertebral column, act upon the column as a whole. The intrinsic muscles, which pass from vertebra to vertebra and act upon the column in segments, and aid in making the whole column rigid for the more general action of the extrinsic muscles. Again, many of the extrinsic muscles, and especially those upon which is usually saddled the responsibility of causing lateral curvature, act upon some distant articulation as well, but the more you examine the subject, the firmer will be your conviction that the muscles implicated are those which pass from vertebra to vertebra. How else can we explain the various phenomena actually found?—and especially the fact that in a group of cases we find no two alike—and why is the curve progressive, involving bone after bone until, sometimes, the whole dorsal region is involved with the frightful deformity characteristic of the disease? The theory that lateral curvative is due to the fact that the right side is the stronger is a fallacy: if any one thing is certain in the pathology of the malady it is this; that in the majority of cases the weakness is on the right side and the contracture on the left and the muscles to be antagonized in the treatment are on the left, the side to which the concavity usually presents. The muscles here form the bow string of the bow and the weaker ones lie over the bend of the bow.

Indeed, I think we may see all the phenomena of lateral curvature in true torticollis. If we look upon the head as a vertebral bone, we see it thrown to one side, rotated, almost rigid in its acquired position and the cause is an almost absolute contracture with ultimate bony changes and accompanied by atrophic disturbances on that side toward which the head is rotated, *i.e.*, the weak or paralyzed side. There is a contracture of the muscle on the shortened side, a lengthening on the weak side, a progressive and permanent deformity of very slow development which would be far more difficult to relieve if the principal muscles affected were so far removed from operative influences as are the small intrinsic muscles of the back—and, as is well known, the etiology of true torticollis is obscure. It begins at such an early age that many writers call it congenital. But the contracture is unilateral; it affects but a few muscles and its early progress is unnoticed, and, indeed, it would go longer unobserved, if the parts involved were endowed with less free movement, or were less exposed to view. Apply these same conditions to the short, deep seated intrinsic muscles of the back and we need not be at a loss to explain all the phenomena of true lateral curvature upon an etiological basis similar to that of true torticollis.

We meet with many cases of simulated lateral curvature caused by pure functional weakness of the extrinsic spinal muscles. Do not fear true lateral curvature in such cases. Simple functional weakness does not produce deformity—and whatever may be the cause of true lateral curvature, functional weakness has nothing to do with it. There is, undoubtedly, a localized or central neural lesion which probably is developed early in life, in this disease. It may be accompanied by acute manifestations which are overlooked or misinterpreted as was the acute lesion which produces the now well known phenomena of poliomyelitis. Whether this be so or not, we know, in the great majority of cases, that there are no acute or subjective symptoms in the first apparent stage of true scoliosis. Whether the cause has been operating for a long time or is of more recent development, the first sign that gives warning of the approach of lateral curvature is either simple, habitual mal-position, or a slightly projecting shoulder-blade, which is detected, not by the patient himself, but by some closely observing friend or the dressmaker. There is no pain unless it be a backache. The patients, especially growing girls, usually present some phase of emotionality, and are, as a rule, of more than average mental activity, and if they do not study very hard, they are expending a great amount of gray matter in useless fretting. They are rarely hysterical in the usually accepted sense of that word—they do not have convulsions, nor are they easily affected as to tears. They are in an almost indescribable "nervous state" which finds almost as many different expressions as there are individuals affected by it, and yet they are all alike in a general sense—still it is so unlike hysteria that I do not regard it as hysterical. I deem it symptomatic of the general condition, for it will be found that this state has existed in a modified form ever since childhood, and simply becomes exaggerated at the time of puberty when all the emotions are intensified.

The signs by which you will recognize true scoliosis have been so fully discussed in speaking of its etiology and pathology, that I shall add only some secondary signs, all dependent upon the primary dorsal curve. These are, first, the position of the shoulders, and, second, the differences in form made by the secondary curves above and below. The position assumed by the shoulders may attract attention almost as soon as the prominent scapula. The shoulder on the side of the convexity is always higher than its fellow—the reason for which is apparent. I call attention to this to say that in those conditions where there is no rotation—the false lateral curvature, there may be a marked difference in the shoulders without the difference in the scapulæ seen in true scoliosis. I do not believe the true disease can occur without rotation; indeed, it is the rotation that really brings to our notice the important objective signs, *i.e.:* the misplaced and prominent scapula. But for this the condition might go on increasing for a longer time without being discovered by the patient's friends, and the fact illustrates how the muscular conditions progress in true scoliosis. If the extrinsic rather than the intrinsic muscles were the principle ones at fault, there would be some gross deformity present as the first symptom—some general deformity, not at that part of the spine where motion is the most limited, but at some part where motion is free—not at that part of the spine where the articular processes permit the most rotation, but at that part where there is the greatest flexibility; not at that part of the vertebral column where the ribs interfere with movement, but where there is no such obstacle to overcome; but the first sign by which we recognize the disease occurs where antero-posterior motion and lateral flexion are the least, and where also the least resistance to rotation is found, and the true curve is limited to this region of the least motion. How can we make the extrinsic muscles responsible for this state of affairs?

The secondary curves give to the spine the so-called serpentine curve. The primary dorsal curve produces a compensatory curve above and another below. If the dorsal curve be to the right, the compensating curves are to the left with what might be called slight half curves at the occiput and sacrum. If the dorsal curve be sharp the secondary curves are marked; if it be gradual, the resultant modifications are so, also. Above, there is the difference between the shoulders and the displaced scapulæ already referred to, while below we find the normal relation of the parts considerably disturbed. There is loss of symmetry between the two sides, there being recession on the side of the convexity and a fulness on the concave side of the dorsal curve. This results in the ''prominent hip,'' so often described by writers, which is wholly due to the primary curvation and the secondary lumbar curve below. It is altogether secondary, and if you are so fortunate as to remove the real curvature it will disappear. Indeed, all the conditions found will give you no trouble if you can successfully antagonize the dorsal curve and the dorsal rotation.

Remembering what I have said, I do not think you will find it difficult to make a diagnosis in what has been called rotary lateral curvature. Indeed, when the curve is marked, no difficulty in diagnosis should occur, but there are some cases presenting in the early stage which are not so easily diagnosed, and I will detain you to call your attention to the means by which you can reach a positive diagnosis. Bear in mind that you cannot have true scoliosis without some rotation and some evident loss of lateral flexibility, due, as before stated, to intrinsic muscular resistance. The rotation is always accompanied by the changes in the position of the scapulæ which I have pointed out. If, with these three conditions present, the rotation, the modified lateral flexibility and the changed position of the scapulæ, and especially if associated with unilateral atrophy of the mammary gland, you will have no doubt. You have what may be called the first apparent stage of true scoliosis. The changes in the scapulæ and mammary gland can be seen at once. The latter is best demonstrated, however, by palpation. You can easily detect, by grasping the mammary glands between the fingers, the difference in size and consistence. But the rotation and the modification of lateral motion are best discovered by tests that I will describe.

In order to detect rotation, place the patient in the erect position, being sure after measurement of the lower extremities that the patient stands upon an even plane. Ask the patient to bend forward as far as possible without bending the knees. This, I believe is Adams' test. If rotation be present, you can readily detect it in this way: you will see it especially in the dorsal region, when the transverse processes will stand out on the convex side and will not be apparent on the concave side. To test the lateral flexibility, place the patient on a stool, in a sitting posture; the stool should equal in height the distance between the sole of your own shoe and the head of the tibia. Seat yourself directly behind the patient upon a chair, place your right elbow upon your knee and the hand upon the ribs—then forcibly, with your left hand put the spine in its extreme lateral position, making a fixed fulcrum of your left knee, elbow and hand. Reverse this process for the opposite side. On that side where the concavity exists (if any be apparent) you will find more movement than on the other—and very frequently, when testing the flexibility of the convex side, you will hear a series of cracks resembling those which many people can produce in the digital articulation at will. This sign is also valuable. When you compress the already slightly wedged articular processes, there is motion at the unaffected articular processes of the other side. When you reverse the proceeding, the wedged processes yield very slightly and frequently with crackling noise. You must be sure of your knee, arm and hand fulcrum though; indeed, I generally throw the knee inward, forcibly, to make the test all the more thorough. The difference in the lateral flexibility of the dorsal region, as shown by this test, is sometimes very surprising, even when the deformity is very slight. Of course you must take into account the general history, age and sex of your patient, the extrinsic spinal muscles, etc., for, while I do not believe these last play any important part in the production of the curve, in the condition that *simulates* true curvature they play the important rôle.

And this brings us to the consideration of differential diagnosis. There is only one condition you are likely to confound with true lateral curvature—and that is the hysterical state which simulates it, and this simulation is sometimes very close indeed. The spine is curved, one of the scapulæ is prominent and there is an awkward position; sometimes, even an habitual mal-position in the erect posture. But though a lateral curve exists, the principle curvature is antero-posterior and partakes of the character of a general excurvation. The scapulæ do not occupy their normal relation to the spine—one being lower than the other, and there may even be the "prominent hip." But there is no rotation in these cases, nor modification of lateral flexibility, and these faults are removed when the patient lies down. The condition is one of functional weakness of the long extrinsic spinal muscles. There is no permanent deformity, the mal-positions change from time to time. It will not be difficult to distinguish the true disease from the simulated one, if you remember that the former presents a permanent, progressive deformity, unaccompanied as a rule by pain, while the latter is a variable condition with pain and "back ache" as a prominent symptom, and that the conditions are removed by recumbency, by manual pressure or by ether. Many of these false curves are called true rotatory, lateral curvature, and are treated as such. Of course they recover: so would they recover, in the majority of cases, if left alone. There are cases in the earlier apparent stage of true lateral curvature when it is sometimes difficult to make a differential diagnosis between the commencing, true, rotatory curve and the false. I have lately covered the point in an essay and refer those interested in the subject to Seguin's *Archives of Medicine* for Dec., 1879, and Feb. and April, 1880. You will there find some facts and conclusions that have greatly helped me in the differential diagnosis of the true and false conditions, not only of the spine, but also of the larger articulations.

True rotatory lateral curvature may exist at almost any part of the spine, though as stated, its usual seat is the dorsal region, and at its upper half. It presents also in the lumbar region, and here, as in the dorsal region, is accompanied by rotation. Some anatomists will tell you that rotation cannot occur in the lumbar region: that the arrangement of the articular processes makes it impossible. We may say, however, it does occur, and I have seen several instances where the primary and inflexible curve was lumbar or dorso-lumbar, accompanied by marked rotation. When, however, the real curve occurs below the 8th or 9th dorsal we do not have atrophy of the mammary glands. The curves below this point, also, are generally not so abrupt as above it, and are not accompanied by such marked mal-position of the scapulæ. The place, at which you may know the real curve exists, is that point at which the rotation is greatest and at which lateral flexibility is most modified. If rotation does not occur at a point that directly involves the scapulæ, there will be no greater mal-position of that bone than is found in cases where there is no rotation. Where-ever the primary curve may be, there you will find, if you search closely enough, the signs by which you will make your diagnosis. In none but the most advanced cases, do you find the normal movements of the other regions modified.

There are five kinds of lateral curvature:—1st, the true curvature, accompanied by rotation:—2nd, the false, or hysterical curve which simulates the 1st:—3rd, the strictly compensatory curve, due to an altered pelvic plane:—4th, those curves that result from acute unilateral pneumonic or pleural inflammation and 5th, the formidable curve that results from infantile paralysis affecting certain spinal muscles. Before proceeding to the treatment, I shall call your attention to the three last named. I might say much more concerning the first two, but time forbids.

The compensatory curves due to causes that tilt the pelvis and which are unaccompanied by any disturbance of muscular equilibrium, are characterized by more or less defined, primary, lumbar curves. If the transverse pelvic plane be at right angles to the antero-posterior median plane of the body, the vertebral column will fall in the latter plane:—but if the angle of the pelvic plane is not a right angle, the vertebral column, will, in the erect posture, vary from the antero-posterior plane to an extent corresponding to the obliquity of the angle of the two planes. Generally speaking, the cause which produces obliquity of this angle is unequal length of the lower extremities which may be either hereditary or acquired. The cause is purely mechanical, and there is no loss of power in the muscles. Again, the position is not a constant one, for the pelvic plane is only temporarily changed during standing or walking. When the patient lies or sits, there is no curvature. These cases never develop true lateral curvature (such is my experience) nor do they require extensive treatment. Equalize the length of the limbs and the trouble disappears.

The next class is peculiar in some respects. The cases depend upon the altered relations which are produced by any cause which results in unilateral collapse of the lung; empyema, pleurisy, pneumonia, etc., may produce this condition. The effect of this state of affairs is this: there results a general curve of the spine toward the healthy side, without any rotation whatever. The spine is drawn, as a whole, toward that lung where respiration is performed, by the extrinsic muscles, some of which aid in respiration. Their antagonists are weakened and some of them do not act; but there is no unilateral action of the intrinsic muscles, and hence there is no rotation. The diagnosis is not difficult; the history points directly to the lung lesion as the cause, and the collapsed chest walls with the physical signs and the general curve will explain all the phenomena. I wish it were as easy to explain the cause of true lateral curvature. The treatment of this class is somewhat difficult; only one lung is performing its function, and apparatus would restrain somewhat the respiratory effort on the unaffected side. I have seen only three or four of these cases and have not used mechanical treatment with any of them. If any apparatus were used, I would employ this, which I am about to show you applied to a case of true lateral curvature.

The curvature resulting from poliomyelitis, like the curvature I have just described, can almost always be easily traced to the central lesion. It, like the other conditions found in infantile paralysis, occurs in early childhood, and in this respect is unlike the true rotatory lateral curvature of adolescents. It is a rare disease, and in all my experience I have seen only two cases. The deformity occurs very early, is slight at first but progresses much more rapidly than the curve of young girls. The same conditions exist as applied to the spine, that are found in infantile paralysis of the lower extremities. The resulting deformity is extreme, and, despite all your efforts, the curvature increases. The amount of rotation seen in these cases is remarkable; it exceeds anything I have ever seen in the adolescent or adult rotatory curve. For in those cases that have come under my observation, there has been undoubted extrinsic as well as intrinsic paralysis, and respiration has been very much impaired. In one case (a boy of eight years) the lesion occurred at two years, the deformity, being to the left, involved, primarily, all the dorsal region. The other patient was a girl. The myelitis also occurred in infancy and the deformity, as you will see by this photograph, was marked, rotation being very great. These cases are very suggestive and several considerations present themselves. First, we have here, occurring in childhood, a deformity which resembles, in all important particulars, that found in true, rotatory lateral curvature. It can be easily traced to an infantile paralysis, which has a known central lesion as its cause. This etiology is, as I said, suggestive. Secondly, so far as we can judge, the principal differences existing between the deformity in the two conditions named are: first, the age at which it occurs; secondly, the rapidity of its progress, and thirdly, the degree of the resulting deformity. Another point is that there is no doubt, in these cases, regarding the part played by the muscles in producing deformity. There is not even a question here of bone lesion as a prime factor. The rigid dorsal curve which results, undoubtedly becomes anchylosed, as all healthy joints would do if maintained for a long time in the same position. But the anchylosis, in this case, is secondary to position caused by muscular contraction. Fourthly, in the case of the girl who was 14 years old and had quite large breasts, the mammary gland on the convex side of the curve was visibly atrophied, and fifthly, there was no doubt in either of these cases regarding the point at which the primary curve existed. There can be no question as to diagnosis in these cases. One was a patient in the private practice of Drs. W. H. Draper and E. C. Seguin, and the patient came to me with the diagnosis made by those gentlemen. The other was also seen by Dr. Seguin, and had extensive paralysis in the lower extremities, and was for several months an inmate of this hospital.

A PRELIMINARY PAPER UPON THE CAUSES OF LATERAL CURVATURE OF THE SPINE; WITH THE REPORT OF A CASE OF LATERAL DEFORMITY DUE TO OCCUPATION.[1]

THE great variety of opinions and theories that exist regarding the etiology and pathology of lateral curvature of the spine, with and without rotation, leads me to place upon record the following case. It presents many interesting points, and illustrates a form of curvature which, I think, has not been fully described, although the causes which produce it, in this individual case, are those most frequently cited by various eminent writers on the subject of scoliosis.

Lateral curvature of the spine has been written upon by so many eminent men, and the subject has been so frequently reviewed, that it may be well, before recording the case that will form the basis of my remarks, to state that I have nowhere seen that lateral curvature has been subjected to the close scrutiny which many other conditions have undergone. I regard this fact as indicative of the lax way in which all strictly objective conditions are regarded. If true scoliosis were accompanied by pain or by extensive destruction of tissue, or by any serious complication, I feel that the causes producing it would have received the attention which they deserve. It is only fifty years ago, for example, that club-foot escaped

[1] *The Archives of Medicine*, Vol. XI, No. 2, April, 1884.

from the realm of the charlatan to the domain of scientific surgery. Within a few years only has knock-knee received the surgical as well as the mechanical attention which it requires, and, although true scoliosis received much attention several years ago, and especially from the French Academy, and many muscles were divided in the attempt to make myotomy answer, in this condition, the purpose of tenotomy in club-foot, the attempt was long since abandoned. True scoliosis, therefore, has little to recommend it to the general or operative surgeon. The requirements of its treatment are almost wholly mechanical, and the most intricate problems in its etiology and pathology still remain unsolved.

Therefore, in dealing with this subject wherein there is so much uncertainty regarding the pathology, and the etiology of which is so obscure, I have thought it well to study the matter from a clinical standpoint until such time as actual post-mortem exploration shall settle certain vexed questions, and, in so doing, I have been able to clearly define eight different varieties of lateral curvature of the spine, each with a distinct etiology, and with easily demonstrated clinical features that should enable the careful observer to differentiate between the several forms.

The varieties are as follows:

1. The true scoliosis, with rotation of the vertebræ.
2. The hysterical imitation of the true curve.
3. The incidental curve due to an unequal length of the lower extremities, etc.
4. The curve due to unilateral lung or pleural disease—first described by Dr. Little, of London.[1]

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[1] *On Spinal Weakness and Spinal Curvatures.* By W. J. Little, M.D. Longmans, Green & Co., London, 1868.

5. The curve, with exaggerated rotation, due to infantile paralysis—first described, I believe, by the writer.[1]

6. The lateral curve, which accompanies the first stage of Pott's disease in the lower and especially the last lumbar vertebræ. To this may be added the lateral deviation that may occur in any case of chronic spondylitis.

7. The rachitic curve; and

8. The curve due to an atonic condition of the fibrous and muscular tissues in the adolescent when prolonged mal-position is maintained, as a matter of occupation especially.

I have had ample opportunities for observation in all of these conditions except the last-named, and the object of this paper is to place upon record a case of lateral curvature due to weakened tissues and habitual mal-position, and to point out the clinical features that characterize that condition.

John L., aged seventeen, born in Ireland and residing in New York, presented for examination at the N. Y. Orthopædic Dispensary on Feb. 4, 1884. The hereditary history shows that his father died of phthisis; his mother is living, and seemed, when the patient was examined, in good health. One sister of the patient has hip-joint disease, one brother is now suffering from inflammatory rheumatism; two brothers are dead from causes unknown. No other facts bearing on heredity could be ascertained.

The patient is a very poorly nourished lad; he has a slight amount of ectropion; his lips are large, thick, and almost colorless; and his nutrition is generally bad. Two years ago, the patient, then fifteen, and apparently straight, entered the service of a grocer in this city as an errand or delivery boy. His employer did not use a horse and wagon for delivery, and all goods delivered were carried by this boy in a basket. The work was laborious and the hours long. In order to facilitate the carrying of the numerous parcels entrusted to him, many of which were very heavy, the patient habitually threw his body forward and to one side, hooking his right arm through the handle of his basket, and supported his burden upon the right ilium; he was in this position a good deal of the time, and, after working steadily for about seventeen months, he began to experience pain in the "right hip" (iliac region) and his friends noticed that he was becoming crooked. As his wages, however, were the chief dependence of his mother's family, he continued his work, disregarding the pain and the increasing mal-position. The pain began to increase, and to trouble him somewhat when he arose in the morning, or upon changing his posture after a period of rest; he found that he could not sleep upon his right side on account of the pain so caused; he began to limp, "favoring" the right leg, and as the pain and mal-position increased, he was at length obliged to abandon his work and to seek medical advice.

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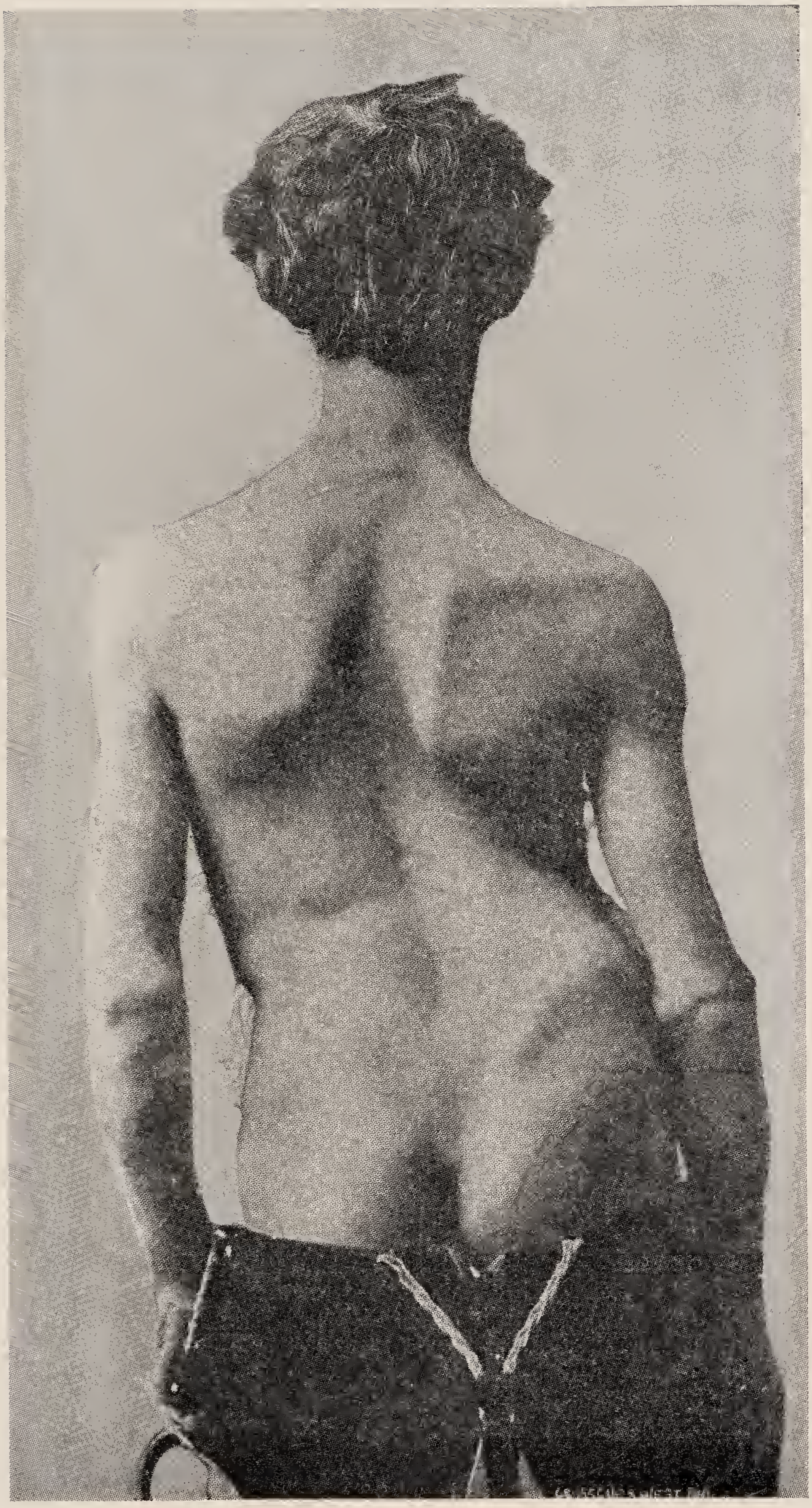
[1] *Pott's Disease, its Pathology and Mechanical Treatment with Remarks on Rotary Lateral Curvature.* By Newton M. Shaffer, M.D., G. P. Putnam's Sons, N. Y., 1879; also, a Lecture on Lateral Curvature of the Spine. By the same author. *N. Y. Medical Gazette*, April 2, 1881.

He first consulted a physician who thought he might have hip-joint disease and referred him to Dr. I. Adler, who eliminated hip disease and sent the patient to the Dispensary. Upon examination, it was found that the pain was referred to several places: 1st, in the back (dorso-lumbar region); 2dly, to the right ilium and especially at the crest, posteriorly, where he had borne his burdens; also in the region of the hip; and 3dly, along the course of the sciatic nerve on the right side. The pain was always "in the back" simply, or "in the hip." None of our inquiries gave rise to any more exact or explicit statement. There were no points of tenderness along the spine; pressure giving negative results. There was no evidence of chronic spondylitis, sacro-iliac disease, or morbus coxarius. The spine was normally flexible antero-posteriorly. There was no marked lateral resistance on either side in the dorsal region, and a pronounced lateral curvature (see engraving) with a very marked prominence of the right ilium, poster-



Lateral Curvature due to Occupation

iorly. The last was a marked feature of the case. On a casual examination it seemed almost as if there were a bony growth at the posterior iliac spine—which impression was strengthened, if anything, by comparison and palpation.

After repeating the tests above mentioned, with the same results I directed the patient to lie down in the prone position. The iliac prominence was then greatly modified, and the spinal curvature much reduced. Gentle traction was then made, one assistant pulling at the axillæ and another at the ankles. After a moment's traction the curvature practically disappeared and the iliac prominence was very greatly reduced. There was no undue rigidity of the spine, as already mentioned, and, as stated, the application of gentle force sufficed to remove all the essential features of deformity.

The patient was then tested as to the peculiarities of the lateral curvature of the spine. The lower extremities were of equal length and this element of mechanical etiology was eliminated. The curve in the lower lumbar region was very short and slightly to the right; it then passed by a long sweep to the left, forming a compensatory curve in the dorsal region. The right shoulder was depressed and there was a slight difference in the scapulæ, the left being somewhat higher. There was a very slight degree of rotation of the dorsal vertebræ toward the left side. Putting the patient to the test of extreme anterior flexion (Adams' test) it became almost straight, and there was then noticed a slight rotation at the lower (lumbar) curve, toward the right side. When the patient was placed in the prone position and traction made, the dorsal and lumbar rotation disappeared. None of these tests gave pain, and there was no evidence of reflex muscular spasm, such as is seen, for example, in chronic spondylitis or in hip-joint disease. Gravity as an etiological factor being removed, the spine, on slight traction, returned in a very brief time to a nearly normal position. All the prominent features of the deformity recurred when the patient reassumed the erect posture. The patellar tendon-reflex was slightly exaggerated on both sides, as were also the others reflexes below the diaphragm. The curve had existed for seven months, during most of which time the patient, with no improvement as to his condition and without modifying his efforts, had continued to work as described above. He was finally compelled to desist by pain and debility, and not by the increasing deformity.

Had this lad been engaged in an occupation that necessitated prolonged standing, instead of walking with his spine distorted, he would probably have developed adolescent knock-knee or flat-foot,—so frequently seen among boys of his age. Be that as it may, he came to us with a deformed spine which presented the features described, and which we have attempted to show in the engraving.

Among the many causes that have been assigned for lateral curvature—the true scoliosis of growing girls with rotation—there is none that is oftener dwelt upon, perhaps, than habitual mal-position. The patient, for example, has been observed to stand habitually, with the weight of the body supported by one limb; or the posture of the subject at the school-desk has been observed as awkward, or somewhat out of normal; and these or similar causes have been accepted as the important etiological factors in true scoliosis. My friend, Dr. W. J. Little, of London, has very recently stated his views upon this subject, and, as they may be accepted as representing a large class of readers and thinkers, it may be well to state them here. Dr. Little says, in his excellent and exhaustive treatise on *The Medical and Surgical Aspects of In-knee* (*Genu Valgum*)," [1] (page 18), speaking of knock-knee: "In adolescents, it is probable that carrying heavy weights, fatigue, and long hours of work have their principal share in its production, favored in fast-growing lads by insufficient diet and consequent weakness of tissue." Again,

[1] London: Longmans, Green & Co., 1882.

page 99, he says: "Nature or gravity works in the lower extremities exactly as in the spinal column afflicted with scoliosis or twisting of the spine, which, we have already stated, is a strictly analogous distortion to in-knee, occurring in distinct forms,—the atonic and rachitic."

The case we have placed upon record would sustain these views, if the history so recorded were the history obtained in true scoliosis with rotation. The present is, however, a very exceptional case in our experience, but, so far as it goes, it corroborates the views of Dr. Little, as above quoted, and no one will deny, I think, that the conditions named by him may produce lateral curvature. Instead of resulting in true scoliosis, however, we have the condition, aptly called by Dr. Ketch, of the Dispensary, "a grocer boy's back."

My friend, Dr. Seguin, has also recently verbally described to me a condition which may be called an "office boy's back," wherein the deformity resulted from carrying bank or other heavy ledgers, etc., and, while it would be impracticable in this paper to point out the differences between this curvature and the other curves, to which we have alluded in our classification, it might be well to state briefly the chief differences between the "grocer boy's back," and that found, for example, in growing girls with rotation.

In my monograph on Pott's disease already referred to, I call attention to the fact that permanent lateral curvature with rotation does not result from inequality in the length of the lower extremities, after such disabling conditions as morbus coxarius, and infantile paralysis of long standing. I have had, since the monograph was written, opportunities to observe many cases of adolescent girls with suppurative hip-joint disease, wherein the affected limbs have been shortened practically from three to five inches or more. In these cases, the prolonged suppuration produced just that atony of the fibrous and muscular tissues that Dr. Little and others place so much stress upon, and the habitual mal-position (also prolonged) was always present when the patients stood or walked. None of these cases have developed anything more than the incidental curve (the third of my classification). All evidences of curvature disappeared in these patients when they were placed in the prone position. Most of them complained, as did John L., of pain somewhere in the back. It has appeared to the writer, therefore, that even atony with prolonged mal-position is not a very frequent cause of true lateral curvature with rotation. The cases are exceptional, I think, wherein these causes produce a permanent curve, *i.e.*, one not obliterated by recumbency, or, as in the case of John L., by recumbency and slight traction.

And this brings us to speak of the principal features in true lateral curvature with rotation. There are many matters that we cannot touch upon on this occasion, and we will refer only to those involved in this case. The first is the permanency of the curve—and the permanency of the rotation, even if slight in true scoliosis. If we place the patient in the position of extreme anterior flexion, with even a slight curve and slight rotation, instructing the patient to attempt to touch the toes with her fingers without bending the knees, the curve remains the same, or is slightly increased, and the rotation is exaggerated. In the case of John L., the lateral element almost wholly disappeared during this test, as did the dorsal rotation. The rotation in the lumbar region, which was slight, and out of all proportion to the curve, became slightly exaggerated, as was to be expected, as this was the point of true curvature, those above being secondary.

If we place a patient with true scoliosis in the prone position, the anterio-posterior curves become modified, as do also the compensatory lateral ones, but the true lateral and rotary elements do not alter even with traction. In other words, we have a structural change in the muscles in true rotary lateral curvature, with, of course, changes in the vertebral bones, which, I think, are secondary. In the case of John L., there is an atonic condition only, without, I believe, structural change in the muscular or fibrous tissues. In true scoliosis there is a permanent, progressive curve, involving especially the *intrinsic* vertebral muscles, while in the case here recorded there seems to be *extrinsic* disturbance only. Ether given to the true scoliotic would fail to modify the curve; if administered to John L. it would have removed the curve at once (inasmuch as recumbency and slight traction did so), though it had existed for seven months or more. In the former condition there is a permanent loss of equilibrium between antagonistic muscles; in the latter the disturbance is almost entirely, if not wholly, functional.

Let me ask those who accept and teach the mechanical etiology of scoliosis, why it is that the curve and twisting occur most frequently in the mid-dorsal region. The thorax forms a pedestal for extensive extrinsic muscular action,—and none of these extrinsic muscles are in a position to produce a thoracic or vertebral deformity, unless their distal extremity becomes a fixed point. Again, there is scarcely any motion at the first five dorsal vertebræ, and the remaining ones—down to the tenth dorsal vertebra—also possess very little mobility. The location of these ten vertebræ, their costal as well as their ligamentous and muscular attachments, the shape of their inter-vertebral discs, their almost interlocking spinous processes, their vertical articular processes, and their situation as regards the centre of gravity (which passes anteriorly to the bodies, in the mid-dorsal region, in the erect position),—all tend to make this the region of the least mobility in the whole spinal column. If the primary curvature were to occur in this region from gravity, would it not be a *posterior* one, as in chronic spondylitis,—this being the normal direction of the spinal curve at this point? And would not the curve occur, if gravity were the factor, at the point of the least resistance, rather than at the point of the greatest resistance—in other words, between the thorax and the pelvis, as it did in the case here recorded? And yet, true scoliosis, with twisting, occurs most frequently in this mid-dorsal region, where there are so many obstacles to its occurrence. To make gravity, with predisposing atonic conditions, accountable for a curve which finds its principal expression at this doubly locked region, seems to the writer wholly untenable.

In the case I have recorded the cause, or causes, are not hard to find. They exactly accord with Dr. Little's graphic description of the cause of adolescent knock-knee. There are many reasons why Dr. Little's etiology, as applied to the development of true scoliosis, should not be accepted. And I have ventured to place the case of John L. upon record as an illustration of this position.

I would like very much to take one step more in this connection, and dwell at length upon what I have come to think are the causes of lateral curvature. I am obliged, however, to desist, both on account of lack of time, and because I am now engaged in making certain tests and in conducting some experiments bearing upon the subject, which are still incomplete. In the lecture already referred to, I have said: "There is, undoubtedly, a localized or central neural lesion, which probably is developed early in life" in this condition. I can only reaffirm this conclusion on this occasion.

POTT'S DISEASE: ITS PATHOLOGY AND MECHANICAL TREATMENT, WITH REMARKS ON ROTARY LATERAL CURVATURE.[1]

PATHOLOGY

THE opportunities for making post-mortem examinations, with a view to ascertain the pathological condition of the vertebral column in the earliest stage of Pott's disease—that is, before deformity occurs—are certainly very rare. If the lesion presented at this period, an *acute* stage, during which the patient might die, our pathological opportunities would be increased, and many points still in dispute regarding its etiology would be definitely settled. But I have never seen, nor obtained the history of, an idiopathic, *acute* spondylitis. That which has ordinarily been described to me as such, I am convinced, has been nothing more than an exacerbation of an insidiously progressive, but semi-latent chronic state. And many of the so-called acute and sub-acute attacks which ultimate in a completely disintegrated joint, are generally found as part of the history of every progressive, chronic, osseous lesion of the articulations. On the other hand, the frequent occurrence of an idiopathic, acute inflammation of some of the structures entering into the formation of the larger articulations, and especially the synovial membrane and fibrous capsule, has suggested to many surgeons that this acute condition may pass by gradual stages into a chronic one. But no one has, so far as I am aware, attempted to trace a chronic spondylitis to a definite, acute, non-traumatic origin. In every sense this disease may be considered a typically chronic one, from its very inception to its ultimate result. Indeed, the disease is so typically chronic, that it is always a difficult matter to decide in each case, as it presents for examination, at just what time the lesion began: nor is it always clear, especially in the latter stages of the chronic lesions of the spine, hip or knee, as to just what tissue was originally attacked.

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[1] G. P. Putnam's Sons, 1879.

If we question intelligent and observing persons who have carefully watched the patient, regarding the early development of the symptoms in a case of Pott's disease, we find, in many instances, that the child has shown an indifference to his ordinary amusements for an indefinite time, which preceded, however, the appearance of pain or deformity. This condition resembles the incipient stage of chronic osteitis affecting the larger articulations, and it points both in chronic epiphysitis of the larger bones of the extremities, and in chronic spondylitis, to a peculiar condition of the system, which, whether hereditary or acquired, places the patient in a state where a slight injury may act as an exciting cause. This peculiar tendency to develop chronic osteitis, either in the vertebral column or the larger epiphyses, upon slight provocation, accounts in great measure, for the readiness with which parents and nurses assign a traumatic origin to the disease.[1]

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[1] Guersant (*Surgical Diseases of Children*, Dunglison's Translation, 1873, page 107), says, "An important observation we may make is, that traumatic arthritis, properly treated, rarely passes into a chronic condition, a fact which we have had many opportunities to verify, although often fearing to the contrary. The cure is assured if the patients are not predisposed to a scrofulous constitution."

I have never had an opportunity to examine, post-mortem, the pathological condition of a vertebral column in the stage alluded to in the first paragraph of this paper, viz., before the appearance of deformity. Even if such examinations were on record, it would require accurate microscopic descriptions of many cases to establish facts regarding the changes which primarily occur, and their most frequent seat.

We have Barwell's conclusions[1] as to the condition of the joint ends in strumous children, which are, I believe, generally accepted as correct. In the absence, among the authors I have consulted, of other important facts bearing upon this point, and based upon actual and very early pathological exploration, I propose, with the aid I have derived from them and from my own experience, both pathological and clinical, to consider the lesion from these standpoints.

Pott's disease, or chronic spondylitis, as I have preferred to call it, may be divided into four stages.

The *first* (the one before alluded to) may be very ill-defined, and easily escape the notice of the careless observer. I saw a case recently, for example, where the mother was certain that the first symptom she noticed was "a pain in the back." This was "a few days ago." The appearance of the child and the deformity did not indicate a disease of long duration, but closer questioning elicited the fact that a few months before the appearance of the pain in the back, he had had "malarial fever." An examination developed an abscess in the sheath of the psoas muscle, which, undoubtedly, was formed during the "malarial fever." A careful examination—without intimating its object to the person most familiar with the history of the patient—will very frequently develop early symptoms which are as insidious, as is the development, in many cases, of the more apparent signs of the disease. This stage may continue for many months, or it may exist for a few days only, and in some cases it is, apparently, absent. Its existence was first brought to my attention by voluntary statements on the part of those having charge of patients, and it may be stated as the result of my own experience in the matter that, in chronic spondylitis, the first symptoms noticed by intelligent people, in the majority of cases, are those which can only be described as occurring in the prodromal stage. I have found that this condition exists with great frequency, and is sufficiently well marked to warrant its introduction as one of the *stages* of the disease.

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[1] Barwell on *Diseases of the Joints*, American edition, page 227.

The *second* stage is that of pain. But this stage, like the first, is sometimes absent, for as Billroth remarks of chronic inflammation of the joints, so clinical experience demonstrates we may say of chronic spondylitis:—"there are cases where the bone is extensively destroyed without any pain."[1] I have frequently seen cases where the kyphotic curve was the only evidence of a progressive, fungous, non-suppurative osteitis of the vertebral bodies. Chronic osteitis of the hip joint also may go on to an actual shortening of the femur, with an entire absence of pain, so far as its *oral* expression is concerned. Pain, however, is present in the great majority of cases of chronic spondylitis, and it is generally accompanied, and not infrequently preceded, by a marked reflex spasm of some of the spinal muscles. This *pain*, which, in the earlier stages of the disease, is most frequently experienced at the periphery of the spinal nerves, probably arises from an irritation of these nerves as they pass through their foramina; but in many cases the inflammatory focus does not in-

[1] Hackley's *Billroth*, page 482.

volve those parts which are immediately contiguous to the foramina, and are we prepared to say that in *all* cases the "gastralgia," so-called, arises from this cause and no other?

The reflex spasm of the muscles does not form the subject of any *voluntary* complaint on the part of the patient. As I first pointed out,[1] this reflex muscular spasm in joint inflammation is due to an irritation, peculiar in its character, of the peripheral nerves which actually supply the diseased or inflamed structure. It is accompanied, however, by an apprehensive feeling which is very difficult to define, but which is certainly very distressing to the patient. This sensation, oftentimes merging into violent paroxysms of pain, is not limited to any particular region, and on account of the intimate relation existing between the minute nerve filaments terminating in the vertebræ, and the great nerve centers, evinces itself in many ways involuntarily. This spasm, which in many cases might be called tetanoid, is present night and day, and after many experiments in diseases of the larger joints, I have found that it will yield to profound anæsthesia only. Certain it is, that the causes which produce this reflex, tetanoid spasm leave upon the patient, after a comparatively short space of time, evidences of severe suffering—evidences which do not follow chronic lesions of the synovial membrane or cartilage.

The *third* stage is that of deformity, and is due to some loss of substance in one or more of the bodies of the vertebræ. Sometimes suppuration ensues rapidly, in others it occurs later, while, not rarely, a morbid process, which has been aptly termed *caries sicca* by German pathologists, occurs, which ultimates in a very pronounced deformity without any evidences whatever of pyogenetic action. When abscess occurs it forms the *fourth* stage. Much might be written regarding this stage of the disease, but the limits of this paper will not permit any extended remarks. I will merely say that the absence of suppuration or any of its symptoms, together with an apparently arrested deformity, not unfrequently serves as a mask to the inherent chronicity of the lesion. Nor does this apparently favorable condition by any means insure the arrest of the destructive inflammation, nor the really progressive kyphosis. There are indications by which the permanent arrest of the morbid action may be demonstrated. But they need not detain us here. The clinical fact to which I wish to invite attention, especially, is that if the symptoms one would naturally expect to find associated with this disease are *not* present, it does not afford positive evidence upon which to make a favorable prognosis. On the contrary, the absence of suppuration after the disease has existed for many months, points, in many cases, and especially in the fungous caries, to an unfavorable prognosis in point of time. The nearer a chronic spondylitis approaches an acute disease in character the more favorable, and of course, the more quickly obtained, is the result. Again quoting Billroth, we may say of spinal caries, as of any other chronic joint lesion,—"a good nutritive condition is the most important point for a favorable prognosis, which would not be very greatly affected by an early and extensive suppuration." An *early* suppuration, even if it be extensive, has, in my own experience, been indicative of a timely and favorable result. In Pott's disease the formation of pus is, as a rule, marked by exacerbations of pain, by an evening rise of temperature, by a peculiar nocturnal cry, which I have named the osteitic, and sometimes—though it is very difficult to obtain this part of the history in children—by slight chilly sensations in the early stage. The rise in temperature, like that which occurs in the suppurative stage of chronic osteitis of the hip or other joints, does not usually exceed 102°–103° in the evening, (I have seen it as high as 105°) and in the morning the mercury drops back to 99°–100°. If the disease occurs in that region of the spine which affords attachment for the *psoas magnus*, this muscle, as well as the *iliacus internus*, may become the seat of reflex spasm. Whenever, in chronic spondylitis, the psoas resists when placed upon the stretch, it is well to explore, by palpation, the pelvic fossæ and the course of the contracted muscle. There are some patients who will, involuntarily, make the abdominal muscles tense when this examination is attempted. Under these circumstances, I administer ether, after the plan of Dr. Packard,[1] of Philadelphia, and, during the primary stage of anæsthesia, which in several of my cases has been brought about in less than sixty seconds, I have been enabled to examine for thirty or forty seconds, without meeting any muscular resistance, the abdomen and pelvic fossæ. In this way I have been enabled to recognize the existence of an abscess at my first examination, and before any external signs whatever of it existed. As it is always important to know whether pus exists or not in these cases, I can confidently recommend this plan, which I first tested in St. Luke's Hospital, with Dr. Andrew H. Smith, in a case where considerable difficulty in making the pelvic and abdominal examinations led to the adoption of this simple, useful procedure.

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[1] "Reflex Muscular Contraction and Atrophy in Chronic Joint Disease," *Archives of Clinical Surgery*, June 15, 1877.

I have attempted to make an intelligent pathological division of the various phenomena of Pott's disease. I cannot avoid recognizing that it is impossible in the present state of our pathological knowledge to do much more than make an arbitrary classification of the disease into different stages.

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[1] *Am. Jour. Med. Sciences*, July, 1877.

In a typical case, we find the stages I have attempted to describe clearly marked. In many cases we find them all present at the first examination: viz., malaise, pain, deformity and suppuration, with no intelligent history as to their sequence. Again, I have seen several cases where profuse suppuration preceded the appearance of deformity, others where paraplegia occurred before the curvature, and many cases where pain, or rather the *oral* expression of pain, was wholly absent, though the kyphosis was marked. I was tempted to make a fifth, or exceptional stage, namely, paraplegia; but this condition is so clearly symptomatic, and, in reality, occurs so rarely under intelligent treatment, that I have classed it among the symptoms of any of the stages. The irregularity, however, with which the various prominent phenomena appear, has also been accompanied by other peculiarities which have added much to the clinical and probable pathological aspects of the subject. They have, in the main, served to strengthen, rather than weaken the division I have made.

As before stated, chronic spondylitis may be divided into two classes, viz., that in which suppuration is a comparatively early symptom, and the non-suppurative, or that in which *caries sicca* occurs. As Billroth forcibly expresses it, "The fungous articular inflammations are often accompanied by caries sicca; the affection may go on for years without the formation of abscess, especially in adults otherwise healthy; there may be extensive destruction in the cartilage and bones, with the consecutive dislocations already mentioned under caries, without a drop of pus."[1] This extremely interesting condition

[1] *Op. cit.*, p. 478.

I have seen very often, clinically, both in the spine and larger joints, and on several occasions after amputation and excision of the larger joints. For reasons which I shall state hereafter, I think it very rare that this condition of caries sicca is seen in the vertebral column after death. Billroth's description of the pathological appearances of this fungous caries is brief and practical. I will condense and quote it here: "If you examine the granulations in the joint and bone, you will find them firmer than usual, and occasionally of cartilaginous consistence, like granulations about to atrophy or cicatrize. Indeed, they do partly atrophy, but the proliferation goes on again, and the bone is destroyed: the process is analogous to cirrhosis; the more luxurious the proliferation of the granulations, the more extensive the destruction."[1]

In the vetebral column, the initial lesion may be, among other conditions, an *osteitis interna fungosa seu granulosa;* an *osteitis interna caseosa;* a *caries superficialis*, following a *periostitis*, or a *caries interna necrotica*. These descriptive titles, taken from different pathologists, express some of the conditions that may be met with clinically, and pathological investigations prove that the original lesion may be termed, in all but the exceptional cases, a chronic osteitis, to which we may add the various qualifying terms to express the exact pathological condition. The German authors use many names to describe the several conditions found in chronic articular lesions, and while subdivisions of the two general classes—fungoid and suppurative (the latter frequently taking the atonic form) may be made with advantage pathologically, the terms *caries* and *chronic osteitis* are used synonymously by many modern writers, the former signification of caries, "ulceration accompanied by suppuration, open ulcers of the bone," being, to a great extent, ignored. It is thus that we speak of a fungous osteitis, where the inflammatory neoplasia proliferate and then contract and cicatrize, but which may degenerate under certain circumstances into an ichorous, flocculent discharge, which marks the atonic form of caries. A very practical remark of Billroth will bear repeating here: "Pathological anatomists who see caries in the dissecting room only, seldom know the granulo-fungous variety well, or regard it the less frequent one. But any one who has frequent opportunities of examining pieces of carious bone during life, especially the resected joints of children, where the process is in a state of active development, will form a very different opinion."[1] The general failure of the vital force which precedes death, produces an effect upon these fungous granulations which results in a rapid breaking down of the neoplastic proliferations of the dry caries—the result is the formation of a degenerate pus, which is discovered at the autopsy. Hence it is that some observers have concluded that "all spinal caries is suppurative."

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[1] *Op. cit.*, page 476.

In a former paragraph I used the phrase, "comparatively early suppuration." I mean by this, when speaking of chronic spondylitis, a suppuration which progresses rapidly enough to appear as a "cold abscess" within, we will say, a year from the first, specific expression of the disease. As a rule, and depending on the site of the initial lesion, the suppurative form of the disease is characterized by the early appearance of pain at the periphery of the irritated spinal nerve, and an abrupt curvature which is apt to result from the rapid loss of bone and fibro-cartilage. Reflex muscular spasm is not so marked in the suppurative as it is in the fungous form—and suppuration, in spinal osteitis as in caries of the larger joints, seems to exert a modifying influence upon the reflex spasm. The peculiar deformity which results, in many cases, from the suppurative form of the lesion, has been for a long time known under the solecism, "Angular Curvature." In the majority of cases, in my own experience, but few vertebræ are involved in this active, suppurative process, though as the disease progresses and involves other vertebræ, the abruptness of the projection may be lost. The caries sicca, on the other hand, more frequently involves a considerable number of the vertebræ primarily, and the resulting deformity partakes more of the character of a true curvature, sometimes, especially in the adult, like the excurvated spine of the old writers.

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[1] Billroth's *Surg. Path.*, Sydenham Ed., Vol. ii., page 158.

In the early history of the dry caries the pain partakes of an apprehensive character, which, however, may become acute under slight traumatic influence, and as in the granular caries of the large articulations, the intense muscular spasm shows the profound impression which the fungous proliferations make upon the nerve filaments distributed to the spongy and vascular osseous tissue. Another fact which I have noticed in connection with these two important conditions—the dry and humid forms of the disease—is that while no portion of the spinal column is exempt from either, the non-suppurative variety occurs more frequently in the dorsal region—more frequently indeed than in the cervical and lumbar regions combined.

The atonic variety of suppurative spondylitis occurs very frequently among strumous children, and is usually described as the typical, strumous Pott's disease. It is, as a rule, accompanied by several of the commonly accepted evidences of scrofula, while on the other hand, I have frequently remarked that the dry caries may exist to a very considerable extent, and progress to marked deformity and loss of osseous tissue, without any marked evidence of dyscrasia in the patient. I have a patient now in my ward at St. Luke's Hospital, whose health is apparently good. There are no evidences whatever of struma, unless we except the joint disease and a slightly tinged sclerotic, and yet he has had a fungoid osteitis of the hip joint for two years. The thigh muscles are atrophied, the limb is shortened an inch, and the joint is practically immobilized by the most intense and persistent reflex muscular spasm. The analogue of this condition in the spine, I have seen very frequently, where all evidences of suppuration were wanting, and the excellent general condition of the patient gave delusive hopes that the kyphos was permanently arrested. This non-suppurative variety of spondylitis, however, is found more frequently, in proportion to the whole number of cases, among adults. It may, however, and following a failure of the general health, it frequently does, assume a suppurative phase. The transformation of the fungoid into the atonic form of the disease is ordinarily attended with urgent symptoms, and in the adult is followed, as a rule, by death. Septicæmia and amyloid degeneration are the most frequent causes of death in the suppurative variety, while tubercular meningitis has only too frequently, in my own experience, followed upon the dry caries, both in public and private practice.

Brodie first called attention to the fact that adults sometimes experience a non-suppurative form of spinal disease. The granular caries was apparently, not known as such to him, and as in many cases, he found a rheumatic history, this eminent writer considered the condition to be of rheumatic origin. In whatever light, however, we view the dry, granulo-fungoid osteitis of the vertebral bodies, clinical experience demonstrates that it is the most tedious disease, either in children or adults, that can affect the articulations: that it is the most difficult to control, and the typical rotary scoliosis alone excepted, the most insidiously progressive.

I have already referred to the reflex muscular spasm, which is not only an early symptom of chronic spondylitis, but also of chronic epiphysitis. This muscular spasm is a prominent symptom of the dry form of spondylitis and when once fairly developed, it is a pretty sure indication that the best efforts in the way of treatment, both constitutional and mechanical, will not suffice to prevent some increase in the spinal curvature. On the other hand, in the more favorable forms of suppurative spondylitis, where the pus approaches the laudable in character, this spasm is not so marked, as I have before stated, and the result, other things being favorable, should be good. Of the atonic variety I need only say that the ichorous, flocculent discharge tells its own story, and that too much should not be expected of an already overburdened constitution.

There are some forms of spinal deformity or curvature, the etiology of which, in the absence of pathological exploration, is quite obscure. I cannot stop here to mention all these conditions, or to attempt to classify them. I may simply say that they seem to have one symptom in common at some stage of their development, viz., a painless contraction or spasm of some of the spinal muscles. In rotary lateral curvature, for example, there is, seemingly, no direct evidence of muscular contraction in the *earliest* stage of the lesion, though I have found resistance to lateral flexion quite marked in many cases. If the intervertebral articulations were the perfect analogues of the larger joints, such conditions might correspond, in their incipient stage, to the chronic synovial inflammations of the latter, which, as I first pointed out,[1] are not accompanied to any great extent by a reflex muscular spasm, and which are also, for a physiological reason, practically painless. I have seen many cases of varied and great deformity of the spine, where the chief agency or factor in the production of the deformity was a painless, but very persistent muscular contraction, and where the apparent condition was, from an ultimate cause yet to be ascertained, an absolute motor paresis of some of the spinal muscles, followed by a shortening of the unbalanced antagonists. In many of these cases also, there are neural symptoms, or even the history of a severe lesion of one of the nerve centers. Such, for example, was the case of H. M., a boy of 14 years. This case was referred to me by Dr. T. M. Markoe, and was seen in consultation with Dr. B. F. Dexter, in November, 1877. In the fall of 1876, the patient, after severe mental exertion at school, had an attack of spinal meningitis, which greatly imperilled his life. He recovered, however, and became, instead of the strong boy he was formerly, a very delicate and effeminate lad. Soon a slight lordosis of the spinal column was noticed. This became slowly progressive, until at the time of the consultation, the anterior curvature of the spinal column was very marked,—resembling indeed the position of the spine in extreme opisthotonos. There was little or no muscular rigidity of the spinal muscles in the erect position. The abdominal muscles were tense; by supporting the abdomen in front, the spine could be gradually straightened, but this the patient could not accomplish unaided. There was no pain whatever after forcible tests. The lordosis disappeared almost completely when the patient assumed a prone position. The mal-position was quite easily corrected with apparatus, but as the anterior curve disappeared under treatment, a typical lateral curvature appeared, accompanied by rotation of the bodies and rigidity, to a considerable extent, in lateral flexion. This is the boy's condition at present, though the scoliosis is under control. There are other cases where, after an insidious prodromal stage, the symptoms partake more of an apparently sub-acute character. The latter stage is generally short, pain being more or less marked. This is followed by a painless reflex spasm of the muscles. The condition described occurs most frequently in the cervical region; or, at least, it is the best studied in the neck, and the muscular phenomena present some very curious and anomalous conditions: for example, a marked contraction of the right sterno-mastoid existing with rotation of the chin to the same side; an apparent anatomical contradiction. This was the condition in a patient, a girl of 12, whom I saw in consultation with Dr. Henry B. Sands in January, 1878, and where we subsequently (February, 1879, one and one-half years after the first symptoms appeared), demonstrated that the muscular contraction was reflex, all muscular resistance disappearing under ether. The lesion in this case was a spondylitis sicca in the upper cervical region, and yet there has been no pain present for over a year, and the patient possesses an apparently normal degree of muscular power in flexing, extending or circumducting the head within the limits of the contraction, just as patients with rotary scoliosis possess, in many instances, a considerable degree of strength in the spinal muscles, the exercise of which does not produce pain. It certainly is a remarkable fact which I have seen demonstrated many times, that a prolonged reflex spasm of certain muscles either of the spine or limbs (yielding wholly under ether) may exist without any history of pain. Another fact, that I have seen too often illustrated to regard as a coincidence, is that in all, or nearly all these cases, the muscular atrophy and spasm could be traced to an osseous lesion, and particularly to the bodies of the vertebræ or the epiphyses.

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[1] *Loc. cit.* and Seguin's *American Clinical Lectures*, Vol. iii, No. VI.

That muscular contractions occur in the typical, rotary scoliosis is very apparent, and that these contractions are reflex, or due to some specific cause, is equally evident when their nature is studied. These cases form, however, a distinct group, yet the ultimate muscular condition reminds me of that which exists in the lower extremities after infantile paralysis. There are other cases where there is a distinct and readily traced paralysis of some of the spinal muscles. The case of H. D. P. will illustrate this condition. The patient was six years old and was placed under my care by Drs. W. H. Draper and E. C. Seguin. The original lesion was a poliomyelitis. Dr. Seguin has furnished me with a memorandum of the muscles primarily affected. Those partially paralyzed (which recovered wholly under Dr. Seguin's treatment) were the muscles of the neck, arm and thigh (left side). Those wholly paralyzed—and which did not recover—were the left *serratus magnus*, the left *transversalis* and *obliquus externus*, and the *supra* and *infra spinati* of the same side. The vertebral column presented an inflexible dorsal curvature toward the paralyzed side, with the usual compensatory (?) curve in the lumbar region. The most marked rotation that I have ever seen occurred in this case, though, as above stated, the boy was only six years old. There was marked contraction of the unparalyzed antagonists on the right side. I have seen several of this class, also, and one patient, a girl of thirteen, is now under observation at the Orthopædic Hospital. I mention these cases and describe their condition, because I am convinced, after studying over three hundred cases of the typical, rotary lateral curvature—and especially the development of the muscular resistance—that no analogous muscular contraction exists, in any condition, without some central or marked reflex cause. At any rate, I am prepared to state that rotary lateral curvature has a specific, pathological cause—not merely a mechanical etiology. The absolute demonstration of this statement and the location of the lesion, of course, lie in post-mortem exploration. On another occasion I shall dwell more particularly upon this interesting and important subject.

I have at least demonstrated from a clinical standpoint that a simple, non-specific loss of muscular equilibrium cannot explain the phenomena of a typical, rotary lateral curvature. In other words, the typical, progressive scoliosis does not develop from a simple alteration of the pelvic plane—where an unequal length of the lower extremities causes, in locomotion, a primary lateral curvature in the lumbar region as a matter of compensation. I have carefully noted the comparative length of the lower extremities in over one hundred cases of the typical, progressive, rotary lateral curvature, and I have found only three where the difference in the length of the limbs amounted to three-eighths of an inch, or more. In view of recent statistics regarding the comparative length of the lower extremities,[1] this, certainly, is a small percentage, as the patients were all either adults or adolescent girls who had nearly attained their growth. On the contrary, where the actual difference between the length of the lower extremities has been great from disease or arrest of development (hip-joint disease, infantile paralysis) amounting to from one to three inches, I have found after examining a large number, only two where the typical scoliosis existed, and, from the histories presented by these cases, I doubt, very much, the etiological value of the altered pelvic plane. In these cases a strictly compensatory curvature results,—but it is readily removed by artificially supplying the necessary length to the shortened member, or by placing the patient in the prone position. In closing this digression, I will merely say that the widest difference exists, clinically, between the purely compensatory curvatures, and the other two classes of lateral curvature to which I have referred. The former are strictly mechanical in their origin, and although, after many years, the intervertebral fibro-cartilages may become modified by the unequal pressure, the curvature is, as a rule, easily remedied in the manner pointed out; while the latter, commencing without any apparent, primary, *mechanical* cause, progresses very insidiously, and, if the progressive deformity be not arrested, it ultimates in a condition which is, of all the abnormal positions which the orthopædic surgeon is called upon to treat, the most difficult to relieve.

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[1] Dr. Cox, *Am. Jour. Med. Sciences*, April, 1875; Dr. Wight, *Arch. Clin. Surgery*, February, 1877; Dr. Hunt, *Am. Jour. Med. Sciences*, January, 1879.

To revert again to the subject more directly under consideration, we may say that in whatever portion of the vertebral column the inflammation most frequently has its origin, it has, as before remarked, many features in common with the chronic forms of disease attacking the larger articulations. The minute structure of the vertebræ need not detain us here, nor is it necessary to enter upon a detailed account of the structure, functions or normal motions of the vertebral column as a whole. The tissues most liable to inflammation are those which interest us most, and we may say that the one most liable to attack is the same in the spine as in the larger articulations. The great joint factors are bone, synovial membrane, cartilage and ligaments. We exclude the fibrous capsule, for while it is frequently the seat, especially in the knee, of acute inflammation, it rarely, in my opinion, becomes the seat of primary, chronic inflammation,—and for another reason—the fibrous capsule is not very extensive in the vertebral column, and is situated at points where the disease rarely has its origin.

We find, if we examine the vertebral bones, that the osseous tissue entering into their structure is of two kinds; the soft, spongy bone existing in the bodies, and the hard, compact tissue being found in the laminæ, pedicles and processes. Synovial membrane exists to a limited extent only, and is not found at points where pathological exploration has demonstrated the lesion most frequently begins. A mass of ligaments bind the bones together, and a series of fibro-cartilages, making, in the aggregate, nearly one-fourth of the entire vertebral column, are interposed between the bodies. Each vertebra, therefore, rests upon a triangular base, the intervertebral disc forming the medium of support anteriorly and the two articular processes posteriorly.

Histologists tell us that the bodies of the vertebræ are very vascular, and Kölliker remarks, "Of the short bones I have found the vertebræ to be the most abundantly supplied with nerves, and especially the bodies."[1] The synovial membrane, while possessing considerable vascularity, has a much less abundant supply of neural tissue, and Kölliker again states, "In the knee I have seen nerves, even in the true synovial membrane, although in general they are rare."[2] Nicolodini[3] also demonstrates by the chloride of gold test the existence of nerves in the "intima" of the synovial membrane. But all histologists that I have, as yet, consulted, agree in stating that the bodies of the vertebræ and the vascular epiphysis have a very abundant supply of nerves, while the true synovial membrane has, comparatively, a very limited supply. The epiphyses are *local nutritive centres*—the synovial membranes are *simple serous membranes.* Morris, in his recent work on the "Anatomy of the Joints," remarks that the synovial membranes "are supplied with nerves and absorbents,"—and in speaking of the ligaments he further says: "Often, however, and especially in the larger joints, it is easy enough to follow nerve branches through the ligamentous fibres of the articulation." The "tendon reflex" would seem to sustain this assertion, though Dr. Gowers, after his recent experiments (*Lancet*, Feb. 1st, 1879), was not assured that the seat of the "tendon reflex" was not in the muscles. It is a well known fact, which scarcely needs repetition here, that articular cartilage, in a normal state, is destitute of both blood-vessels and nerves.

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[1] *Manual of Human Histology*, Syd. Ed., Vol. i, page 336.

[2] *Ibid.*, page 338.

[3] *Stricker's Jahrbucher*, 1873.

The intervertebral discs, according, to Kölliker and others, consist, 1st, of exterior concentric layers of fibro-cartilage and whitish connective tissue; 2dly, of a central, principally, fibro-cartilaginous substance; and 3dly, of two cartilaginous layers applied immediately to the bones. The soft, central substance does not differ materially in its elements from cartilage, for the microscope shows the preponderance of fibro-cartilage cells. These discs contain, like the true articular cartilage, neither vessels nor nerves, in a normal state, but vessels may, under certain morbid conditions, develop in them (Rokitansky, Kölliker). They may also undergo two species of degeneration, viz., atrophy and ossification. The former occurs from very obscure causes, and is accompanied by no marked symptoms. These two conditions are found principally in senile spinal curvatures, though Rokitansky states, "true ossification of fibro-cartilage is, in every case, highly problematical."[1] The same authority states, "Inflammation though rarely met with in the fibro-cartilage, does unquestionably occur in them. It is remarkable for its acute course, and for the rapid ulcerative destruction of the fibro-cartilage to which it leads. An inflammation is sometimes met with in the intervertebral cartilages, which terminates sooner or later in suppuration, and is generally combined in the end with inflammation and caries of the bodies of the vertebræ."[2]

My own experience leads me to believe that an *acute* inflammation of either the intervertebral discs, or of the ligaments, would find expression in considerable pain. We know, for instance, that an *acute* synovitis of the knee-joint is exquisitely painful, while a *chronic* synovitis may exist for years without any expression of pain at all. Acute inflammation of any of the appendages of the vertebral bones (excluding those due to direct traumatism) are quite rare, and I have seen only a few cases which could be thus classified. Two of them, I am inclined to believe, were lesions of the intervertebral cartilages, with suppuration. Markedly acute symptoms, high temperature, the rapid formation of pus, and complete recovery in a few weeks with slight deformity and rigidity of the spine, limited to two or three vertebræ, summarize their histories. Primary inflammation of the ligaments, as a factor of chronic spondylitis, I do not think, occurs. Their structure would not predispose them to a chronic form of inflammation. For evident reasons the spinal synovial membrane should not give rise to much trouble, and Brodie, after many post-mortem examinations, says,

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[1] *Rokitansky's Path. Anat.*, Syd. Ed., Vol. iii, page 281.

[2] *Ibid.*, page 279.

"Although it is not to be supposed that the synovial membrane belonging to the joints between the articulating processes of the vertebræ are altogether exempt from the liability to inflammation, there is no doubt that inflammation in them is of rare occurrence, and no case has fallen under my own observation in which the existence of such disease was proved by the examination of the dead body."[1]

I have analyzed thus briefly the histology and pathology of the important joint factors, so far as the authorities I have referred to throw direct light upon the subject, I might cite others also, were such a course necessary, in confirmation. I have also summarized their pathology in the light of a very considerable experience, based upon a service of over fifteen years in public institutions especially devoted to the treatment of deformities, and notably those of the spine and hip. It is, however, to be regretted, that there exist almost insuperable difficulties in the way of obtaining autopsies in dispensary and hospital practice, especially among children. I not only find that many mothers object to leaving their children in any hospital ward, but that the bare suggestion of an autopsy is sufficient to incur the lasting displeasure of the friends of the patient. In dispensary practice patients that are seized with an acute, intercurrent trouble, usually find their way to some general dispensary for treatment, and if death ensues, we are made aware of the fact only when they are beyond the reach of post mortem examination.[2] Even if these facts were otherwise, the post mortem appearances of advanced Pott's disease are not always satisfactory, for reasons already stated. I have therefore found my greatest pathological aids from examinations of the diseased tissues in the larger joints after resection and amputation. We cannot observe the spinal column under these favorable conditions. As Dr. Heitzman expresses it in his lectures on the "Development of Bone," when referring to the comparative value of microscopic examinations of recent and dried specimens of osseous tissue, ". . . it is the difference between examining a mummy, and the recently deceased body." In stating the matter in this way, I have no wish to underrate the contributions of others, or to expose myself to the criticism of discarding the evidence obtained at autopsies made in advanced Pott's disease. Compared with these latter evidences, however, I regard the fresh specimens examined immediately after resection or amputation, as being far more valuable than those obtained after the changes have occurred which precede and follow death.

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[1] *Diseases of the Joints*, 5th Ed., page 300.

[2] Of 299 cases of Pott's disease treated at the Orthopædic Dispensary in 1877, 10 died. One from Bright's disease, 2 tubercular meningitis, 1 whooping cough, 3 pneumonia, 1 diphtheria, and 2 unknown. During 1878, 267 patients were treated for Pott's disease in the same institution. The deaths were 13; 2 Bright's disease, 1 tubercular meningitis, 1 bronchitis, 2 scarlitina, 2 myelitis, 2 phthisis, 1 exhaustion, and 2 unknown.

Viewed in the light of the symptoms presented, the pathology of chronic spondylitis becomes more interesting, and many important conclusions may be drawn by applying the known histological and pathological facts, to the indices of this insidiously progressive lesion. The most important symptom upon which we are obliged to rely for the early diagnosis of certain forms (the osteitic) of articular disease and before an actual loss of substance occasions deformity, are those which come to us through the medium of the nervous system. It is a remarkable clinical fact to which, so far as I am aware, I was the first to call attention, that a *chronic* disease may exist in any given articular tissue which is sparsely supplied with nerves, or which is devoid of neural elements, without presenting any *subjective* symptoms which are of sufficient importance to attract the attention of the patient. Instances of this may be found in the chronic lesions of the synovial membrane, and the cartilages of the larger articulations. Atrophy and almost complete disappearance of the cartilage may occur without any symptom during life, as before stated, and among the many instances of chronic synovial inflammations which I might cite, I will briefly state the case of a young lady of 22 years, who recently consulted me regarding a diseased knee. The history of the case covered ten years, and the first symptom noticed was a simple, painless swelling of the joint. Local rise of temperature occurred later and has been present most of the time, but the important fact that I wish to call attention to is, that during the past year only, has there been any pain. Since the bone has become involved in the diseased process which originated years ago in the synovial membrane, there has been limping (there was none before, except after very long and fatiguing walks), nocturnal pain, "starting of the limb," contraction of the joint, and a more rapid atrophy of the muscles. When the bone becomes involved, the neural response is unmistakable. The marked reflex spasm of the muscles occurs quite early in all inflammations involving the epiphysis or bodies of the vertebræ, and is almost uniformly absent, or is developed only to a slight extent, in all true, chronic synovial inflammations. It has become an interesting matter in this connection to study the origin of the nerves which supply the vertebral bodies. It has been demonstrated by Kölliker, Luschka and Kobelt, that the nerves which terminate in the vertebral bodies can be traced not only to the cerebral and spinal nerves, but also to the great sympathetic. In this way does the irritation of the peripheral nerves supplying not only the vertebral bodies, but also the larger epiphyses (for the same thing has been demonstrated in connection with them) give expression to such a variety of neural manifestations, viz., pain (which may or may not find expression orally); a peculiar, persistent and involuntary muscular spasm; a pronounced and direct atrophy of the muscles thus affected; an agonizing and piercing nocturnal cry, (which, however, may be present only for a limited time), and a general, particularly a facial, expression of suffering not always seen in the more especially fatal chronic diseases of other tissues. In the same way may be explained the absence of these symptoms where a tissue with a sparse neural basis[1] is attacked with the same chronic inflammation. In other words these simple facts explain many important differences which exist between chronic osteitis and chronic synovitis, and they form valuable aids in the differential diagnosis of these two conditions, as I have pointed out in the Clinical Lecture before mentioned.

The primary lesion in Pott's Disease gives marked evidence, in all but exceptional cases, of these early and marked neural symptoms. In chronic disease of the knee-joint, for instance, we look for our first symptoms to the bone or synovial membrane. The latter present only, at first, *objective signs*—without any important *subjective symptoms.* In the spine we must look to the bone for our first symptoms, for the intervertebral discs, like the synovial membrane in the hip-joint, may, I believe, continue in a state of degeneration for a long time without giving any important evidence of the fact. The cartilage becomes primarily affected, in exceptional cases only. Those cases in which I have been able to diagnose an *acute*, fibro-chondritis, with suppuration, the patients have recovered without any deformity other than a slight irregularity, with rigidity, limited to two or three vertebræ, as before stated. I can imagine, however, that the intervertebral fibro-cartilage may become diseased without involving the bone. Brodie states[1] that he has observed ulceration of the intervertebral cartilage where the bones were "in a perfectly healthy state." I can understand how a central osteitis may occur in a vertebral body without producing disintegration of the disc. But, other things being equal, and the fibro-cartilage depending for its nourishment upon the bone, it is very exceptional that the initial lesion of either Pott's disease, or chronic osteitis of the knee-joint, for instance, occurs in the cartilage. In any event, in chronic joint affections, until the bone is involved, the marked neural symptoms do not occur, and I may repeat here the conclusion I reached in my study of the "Etiology and Pathology of Chronic Joint Disease," viz.: that "*reflex muscular spasm in chronic joint disease always indicates osteitis.*" That this muscular spasm is always present, to a greater or less extent in chronic osteitis affecting either the articular ends, or the bodies of the vertebræ, I am assured. Its presence is due to a direct pathological cause and is not "conservative"—a fact which I have taught for the last two or three years at the Orthopædic Dispensary—and was, I believe, the first to point out. This pathological, yet local, muscular spasm is easily explained, and its purely reflex character may be demonstrated by the administration of an anæsthetic—when, if the muscle has not undergone structural change, the spasm will wholly disappear, and the "fibrous anchylosis" gives way to a perfectly movable joint. In the early history of the spasm, the patient can voluntarily exercise the affected muscles within certain limits, as is illustrated in the case of spondylitis sicca seen with Dr. Sands; but in the latter stages, the muscular spasm does not permit the patient either to flex or extend the joint. The muscles become, in the first stage, temporarily contracted under the stimulus of extreme motion or pressure, and in the last stage, this spasm may so fix the joint that no motion can be detected by the most delicate examination, and this long before the muscles become structurally shortened.

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[1] It is probable that the great difference existing between the symptoms of chronic osteitis and chronic synovitis which are here pointed out, arises not only from the inequality of the neural distribution to these two important joint factors, but also from the fact that the synovial membrane being a simple tissue, like other serous membranes, and not a local nutritive centre, like the epiphyses, does not possess such profound and intimate relations with the great nerve centres.

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[1] *Op. cit.* 5th Ed., p. 301.

After all, viewed in the light of their symptoms there are but few important differences between a chronic spondylitis, and a chronic epiphysitis of one of the larger articulations. The pain at the periphery of the spinal nerves—first noticed by Brodie—has its analogue in the pain at the knee in hip-joint disease. The reflex muscular spasm which renders the vertebral column, at the point of disease, very rigid, also finds expression in the same localized, muscular spasm which occurs in coxo-femoral osteitis. The hip joint is not only held rigid by the muscular spasm, but the various positions assumed by the thigh, producing the deformities in joint disease, are due to the same cause. In some other respects the two conditions resemble each other, but enough has been said regarding the similarity of these two pathological states. That the symptoms are so nearly allied is, at least, corroborative evidence that, in chronic disease of the articulations of the lower extremities, the lesion most frequently to be diagnosed is a *chronic osteitis*, and not a *chronic synovitis*.

Several practical deductions based upon many experiments and observations of the phenomena of chronic diseases of the hip and knee, may be of value here as bearing upon the mechanical treatment, which I shall soon consider, of Pott's disease. 1st. A severe examination of a diseased joint by making forcible and extensive movements of the articulation, under ether, is almost always followed by a decided aggravation of the symptoms; 2dly; If ether be administered with a view to perform tenotomy of the contracted muscles, and the contraction disappears after the anæsthesia becomes profound, the mere division of the tendons will accomplish but little, for the spasm will recur as soon as the reflex power is recovered, and the complete reunion of the tendon is a question of a few days only; 3dly; So long as the joint movements are impeded by this localized, reflex muscular spasm, during consciousness, and all muscular resistance disappears under ether, an osteitis of, or near, the articulation exists; 4thly; As in chronic epiphysitis, so in chronic spondylitis, the reflex, tetanoid spasm yields to ether, but not to such agents as morphine and chloral, if administered in the customary doses.

When suspension is employed now-a-days "to reduce the deformity" of Pott's disease, it cannot, for reasons to be assigned hereafter, do more than modify the compensatory curves, unless, ether be administered after the plan of the German surgeons. If this be done—and it cannot, in my opinion, be other than dangerous, or at best, useless—it will be found that the pathological curvature is more readily reduced, inasmuch as the reflex spasm yields when an anæsthetic is administered. It is due to the presence of this muscular spasm, during the process of suspension without an anæsthetic, that more harm is not done by the rejuvenated custom of using "the gallows" in the treatment of Pott's disease.

TREATMENT

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I have been fortunate enough to obtain a very considerable number of works upon spinal deformities. A careful perusal of them leads me to express surprise that so little attention has been paid by the vast majority of their authors to the actual pathology of the diseases which produced the deformities. Had the pathology of the different lesions been studied more carefully before the treatment was devised, there would have been far fewer "machines" and much less suffering would have been inflicted by the various, crude appliances that are pictured in the works of many writers, even of recent date. It has been the habit of many of these authors to ignore the pathology, and to then "devise" a mechanical treatment. They have apparently looked upon the distorted spine as a mechanic would contemplate a curved, metallic bar, which needed only a certain degree of force to straighten it. But, as the spines are bent in obedience to a mechanical law—of course—but not from a primary mechanical cause, the curvatures, in many instances, went on progressively just as if they had not been treated from an empirical standpoint. And herein, in my opinion, lies not only the error of the past, so far as the scientific treatment of spinal curvatures is concerned, but also the danger of the future. It is a proposition which, I think, can be easily demonstrated, that errors of form which do not involve vital parts, and which are due to, strictly speaking, mechanical causes alone, need, as a rule, mechanical treatment only. But the deformities of the spine having their origin in the lesion which produces Pott's disease, and in the primary change which ultimates in the typical and progressive rotary lateral curvature, have many elements which cannot, I am convinced, be explained upon any mechanical or traumatic basis. We are apt in Orthopædy, as in other departments of Medicine to confound *cause* and *effect*, and thus a series of etiological errors have arisen, based upon mechanics and traumatism, which cannot be too soon removed. Years of study and exploration will probably pass before many of these errors can be removed. In the meantime various opinions will be held;—the human body will be looked upon, by some, as a simple series of mechanical actions, and "machines" will be made with the object of accomplishing the impossible. Mechanical "demonstrations" of the etiology of lateral curvature, for instance, illustrated in the dried and mounted vertebral column, and arranged with obedient springs and checks, cannot represent the curving spine with its multiplicity of functions and levers, and the vital force controlling and operating them;—nor can the progressive deformity of Pott's disease be explained upon the basis of traumatism and gravity. The agencies at work in the production of spinal curvatures—whether lateral or posterior—would operate, I am convinced, in many cases, were both these elements removed; and while *they both become very important factors after the lesion is established*, we must look further and deeper for our primary cause.

In entering directly upon the treatment of Pott's disease, I shall avail myself, as I did in studying its pathology, of my experience in treating analogous inflammations of the larger articulations. There are so many points in common, as relating to the etiology, pathology and symptoms of these two conditions of chronic spondylitis and chronic epiphysitis in children, that it would be difficult to consider the one condition, in the present state of our knowledge of these diseases, without drawing conclusions based upon our observations, both clinical and pathological, of the other.

The difficulties in the way of successfully treating Pott's disease mechanically are, sometimes, very great. If we were dealing with a simple traumatism as a cause, and gravity as a second factor, the mechanical treatment of Pott's disease would present scarcely greater difficulties than the mechanical treatment of fracture, for instance. Unfortunately, as I have pointed out when considering the pathology of this lesion, we have to deal with a very insidiously progressive disease, where very important factors in the production of the increasing deformity have been either ignored or overlooked by those who claim that the lesion has a traumatic etiology. And I may also say that many of those who have regarded the disease from a constitutional standpoint have also disregarded certain pathological conditions which contribute to the progressive character of the disease. A progressive inflammation, involving a gradual loss of substance—whether by a process of "regressive metamorphosis," which ultimates in a flocculent discharge, or a fungous proliferation unaccompanied by suppuration, and occurring in such a tissue as we have found the vertebral bodies and the epiphyses to be,—must, it seems to me, be accompanied by peculiar manifestations. I have tried to point out what some of these manifestations are, and to trace their relation to the lesion. It is the pathological value of these symptoms to which I wish to call attention among other matters, in pointing out the mechanical difficulties which present in the treatment of chronic spondylitis.

And first let me ask, is there any mechanical treatment which can wholly cover the pathological condition met with in a typical case of Pott's disease? Can we expect any mechanical treatment to always, or even as a rule, cure a progressive lesion of such magnitude as that we have attempted to describe? Are we justified in claiming that we can accomplish any more, by an artificial mechanical appliance, in an osseous disease where motion acts as a great factor in aggravating the symptoms, and where the lesion exists in a tissue especially endowed with vessels and nerves, than we can in a somewhat analogous pathological condition in the shaft of a long bone when the mechanical treatment is supplied by nature herself? There are those who claim that we can. I differ from the gentlemen who make the assertion that Pott's disease is traumatic in its origin, and hence quite easily cured, and I do not deem an apology necessary in this connection for the statement that my conclusions are founded upon an experience which is based upon an aggregate of many thousand cases of joint and spinal disease, the histories of over four thousand of which were either recorded by myself, or taken under my personal instruction.

What then should be the indications for the mechanical treatment, for all will admit, in any event, that the elements of locomotive traumatism and gravity, after the lesion is established, make mechanical treatment of some kind absolutely necessary? Perhaps I can better answer this question, after experiments directly bearing upon this point, by stating what they should *not* be. 1st. Mechanical treatment, either in the spine or larger joints should not be used with the idea of overcoming the muscular resistance. We may succeed in antagonizing the muscular spasm to a certain extent, but it cannot be annulled by any mechanical therapeutics we may devise. As I have previously stated, neither morphine nor chloral, even in quite large doses, perceptibly modifies this tetanoid spasm in a well developed case. 2dly. The traumatism of suspension, like the traumatism of forcible examination in articular osteitis of the hip, has in my own experience, in some cases, aggravated the lesion, increased the subsequent muscular resistance and intensified the pain. I have seen irreparable damage inflicted by an unnecessarily severe examination of a suspected hip joint under ether, and I have known more than one case where the error was made of eliminating any joint lesion because "stiffness of the joint" relaxed under an anæsthetic. Even more—I have had cases under my care, where the surgeon previously in charge of the case had diagnosed an "hysterical joint," a sciatica or a rheumatism—for the same reason. It is a fact which cannot be mentioned too often, *that the reflex spasm of the muscles in chronic osteitis of the larger articulations will yield to anæsthetics only, and that the anæsthesia produced by ether or chloroform suspends all the really important symptoms upon which we should rely for our diagnosis, especially in the early stages of the lesion.* Hence, as a rule, ether should not be administered in joint disease for diagnostic purposes, except to eliminate doubtful points, and for the purpose of ascertaining the condition of a joint with reference to exsection. Some German surgeons have recently administered anæsthetics in cases of Pott's disease, prior to the application of the plaster jacket. Under these circumstances it is found to be a comparatively easy matter to reduce the pathological curvature. We see here, again, the effect of anæsthesia upon the muscular spasm, for, without ether, I have never seen the true pathological curve reduced by suspension. This procedure, which I have often wished to test but never cared, for obvious reasons, to attempt, has determined a question which would still remain unsettled without some such demonstration. It proves that the weight of the body, acting as a counter extending force during suspension, is not sufficient to modify the curvature due to the lesion itself, while if, under the same circumstances, ether be administered, the projection is more easily reduced. If, in the first instance, the kyphosis remained unchanged from adhesions which had taken place, it would have remained so under ether, other conditions, of course, being equal, and the modification of the true curvature under anæsthesia demonstrates, to my mind, with a multitude of similar experiences in the hip and knee, that the reflex muscular spasm is as important a symptom in chronic spondylitis as it is in chronic epiphysitis, and that it bears the same relation to the lesion.

With these preliminary remarks, I will proceed to detail the results of my experience in the mechanical treatment of Pott's disease, and to consider the two most prominent methods now before the profession.

The mechanical treatment of the two conditions most frequently mentioned in this essay, viz., Pott's disease and hip-joint disease, varies with the widely different functions performed by the structures involved. In hip disease, for instance, we have a lesion involving only one articulation, and as, anatomically, there are excellent opportunities to make both extension and counter-extension, we can so apply our force against the tuber ischii as to produce that kind of fixation which relieves much of the pain, antagonizes, to a greater or less extent, the muscular spasm, and places the joint under the best local condition for repair. But the spine does not offer any anatomical facilities to apply such a method of treatment, and it is absolutely impossible to apply a continuous extension and counter-extension which can be maintained with any degree of success. It would be an especially difficult task to so direct the extension that the ultimate force would be expended upon the diseased bones alone. Let us suppose, for instance, that six or eight bones are diseased, the intervertebral discs disintegrated and the ligaments badly implicated, as is not unfrequently the case, how could we be assured that the extension reaches all the diseased tract? When extension by means of suspension is applied, as it frequently is now-a-days, to the whole vertebral column from the first cervical vertebra down, in cases of spinal disease and the curvatures resulting therefrom, how much of the apparent change that takes place in the projection is due to the effect produced upon the projection itself? It is a well-known fact that our height is increased in the morning after a few hours' rest in the recumbent position. Extension made through the healthy intervertebral fibro-cartilages and the other structures binding the vertebral bones together, for a few moments only, is capable of lengthening the vertebral column to a very considerable extent. How much one-half hour's extension or suspension would stretch these healthy tissues I do not know. But when the extension is applied to the healthy spine the normal curves are also obliterated and the spinal column becomes straight, as it is in early infancy. When the same force is applied where a portion of the vertebral column is diseased, the compensatory curves which result from the changed centre of gravity are also greatly modified, and the deformity is thus placed under far different relations to the healthy parts of the spine, and without, in my own experience, affecting, to any appreciable extent, the true, pathological curvature. In other words, the change is apparent rather than real, and the great increase in height noticed after a suspension of this kind, is due to the extensibility of the unaffected structures, and the obliteration or modification of the compensatory curves. There can be no actual lengthening of the vertebral column except that which comes from the elastic nature of the ligaments and fibro-cartilage. If, after applying suspension, the projection should decrease it is due to the separation of the diseased osseous surfaces at the expense of pressure upon the articular processes—making a lever of them, in other words, the weight of the body below forming the resistance, and the cervical region being the seat of the application of the suspending power. If suspension separates the bones, it does so at the risk of breaking up any reparative process that may have begun, and I am inclined to think that it may accomplish more, in this respect, than is advisable in advanced cases, and if this same force be used, the tetanoid spasm of the muscles prevents, in a great measure, separation of the diseased surfaces in the more recent cases.[1]

Recumbency, in the prone position for a few moments, will produce all the "separation" necessary; indeed, all the "separation" required is that which produces an adequate modification of the injurious pressure or contact. This gained, we have accomplished just as much, as a matter of treatment, as if we had actually drawn the bones asunder, and the gap thus created by, what must necessarily be, forcible means, cannot, I am convinced, be filled by osteophytic action.

I have seen cases where the projection apparently decreased under the extension produced by suspension.[2] I confess that in these cases I have proceeded very carefully with the plastic envelope. But these patients have done no better than others. My experiments have proved to me that the weight of the body below the point of disease could not overcome the reflex muscular spasm which resists our mechanical efforts to overcome it. It is, probably, very fortunate that this is so, for even if the spine could be straightened it would lead to false hopes of a speedy cure; and what right have we, even if continuous extension and counter extension could be accurately maintained, as in cases of hip- and knee-joint disease, to expect that we could cure spinal caries any more rapidly than either of these former conditions, which, even under favorable circumstances, may take years to accomplish? Another fact I have noticed is, that when patients return for a second application of the plaster jacket, I have found, before the old apparatus was removed and suspension again applied, that the spine had fallen back, to its old position, or nearly so. It is certain that a comprehensive mechanical extension in a typical case of dry osteitis of the hip or knee joint does no more than to slightly modify the reflex muscular spasm.[1] Suspension or extension, even if they could be made continuous, can do no more in Pott's disease. Some years ago it was the custom to treat hip-joint disease by applying a plaster of Paris splint, after extending the limb. This plan was thoroughly tried, found useless and abandoned. It would be as scientific, in the present state of our knowledge of the pathology of hip-joint disease, to suspend the patient by

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[1] If we place a patient with hip disease upon a soft mattress in the supine position, and after flexing the thigh and grasping the pelvis, attempt rotation of the hip joint, we find, in many instances, that little or no motion can be developed in this direction. If the case be one of caries sicca, I find it happens very frequently that, after having made this test, the limb be allowed to lie quietly upon the mattress for a few moments, quite a degree of rotation can then be developed, if the entire limb be allowed to rest upon the bed, and only a gentle force be applied at the knee.

[2] I am convinced that this apparent decrease arose from the fact that the compensatory curves were so great, producing such a marked effect when suspension was used, as to leave the impression that the pathological curvature had been actually reduced. If only two vertebræ are diseased, for instance, the resulting posterior curvature may seem to involve the three or

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four contiguous bones above and below. It is especially under these circumstances that I have found such marked changes without affecting, in reality, the real projection.

[1] That "extension with motion" is a fallacy in the mechanical treatment of *chronic osteitis* of the hip or knee, is very easily demonstrated.

the leg while we put a plaster jacket on his hip, as to expect, by simply suspending the patient by the upper vertebræ, making him (we will say), one or two inches taller at the expense of healthy tissues and the modification of the compensatory curves, and then permitting the spine to gradually sink back into its old position, we could cure spinal caries more rapidly than by a more reasonable and a more scientific method.[1]

To those who have had a considerable experience in the treatment of chronic articular diseases, and more especially Pott's disease, I am, I know, expressing a very common experience when I say that the application of almost any support which acts upon correct principles, affords almost instantaneous relief. In some cases the primary effects are almost magical, and are apt to mislead those to whom the scientific, mechanical treatment of articular diseases is a novelty. In dispensary and hospital experience—as well as in private practice—I have frequently had occasion to remark this fact. But relieving the symptoms and curing the disease are two very different matters. Treatment by the gypsum bandage forms no exception to the rule which applies to the other forms of apparatus—or, indeed, which applies to any local measures adapted to the treatment of a constitutional disease. The same may be said of the actual cautery in Pott's disease—it affords temporary relief in many cases—but it cannot be called curative any more than any other form of efficient counter irritation. It has been my experience, for instance, to apply a plaster jacket to a patient—a little boy (not the one referred to in the last foot note) in my service at St. Luke's Hospital. It was, apparently, a favorable case, and one where a good result might be anticipated. The disease was in the lower dorsal. His breathing became better after the jacket was applied, pain was relieved, the patient walked with a firmer tread, and, to the uninitiated, the result promised brilliantly. I had seen too many other cases, however, of similar character treated by various methods, which progressed in the same satisfactory manner for awhile, to expect too much of any local treatment. The patient died in a few months from amyloid degeneration. I have seen many cases of Pott's disease, promising cases too, improve at first very markedly under Taylor's antero-posterior support,[1] only to find in a few weeks that the *psoas magnus* was contracting as a premonitory symptom of abscess, or some other complication presented which would cause those who might formulate a brilliant prognosis in the early history of the mechanical treatment, to very materially modify their opinion. This much may be stated as the result of my clinical experience,—suspension, with the application of the plaster jacket is not so likely to cure caries of the vertebræ as would the same methods applied to caries of the larger joints. The fact is that, after a chronic disease has been once set up in a vertebral body or an epiphysis, there is nothing that aggravates the morbid process, or produces pain and suffering, more, than the traumatism which results from the movement and pressure which is imparted by locomotion. It not unfrequently happens that about all that *any* apparatus accomplishes is to prevent this traumatism, and in this way to modify, or greatly mitigate, the symptoms. So in Pott's disease, apparatus rightly constructed, be it of steel, or plaster, prevents motion at the point of disease. But the disease still exists, and although the apparatus places it under conditions favorable to recovery, the result, not unfrequently, is beyond our control. If Pott's disease were traumatic in its origin, as many seem inclined to believe, no doubt a speedy cure would result under any comprehensive local treatment embodying correct principles.

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[1] A case now in my service at St. Luke's Hospital will illustrate some of these points. The patient, a boy of 6 years, has Pott's disease in the dorso-lumbar region, involving four vertebræ. In the course of five months I applied three plaster jackets, carefully and snugly, after suspension. When the first jacket was removed, the projection was evidently larger, and this was the case after each of the others had been taken off, until, when the last one was removed, the deformity had increased nearly ¾ of an inch. The patient was carefully watched, and I personally inspected the jacket several times a week during the treatment.

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[1] To avoid repetition and ambiguity, I will here state that when the phrase "antero-posterior support" is used in the subsequent pages, that form of apparatus which embodies the principles of the "Taylor's spinal assistant," or the "Davis spinal brace" is indicated, unless otherwise specified. In like manner, "plaster jacket" refers to the method and apparatus which were first used by Dr. Bryan in the treatment of Pott's disease, and subsequently introduced to the profession by Dr. Sayre.

I would say a few words more regarding the great relief that follows the use of a properly adjusted apparatus in Pott's disease. It is so marked in many cases, that the friends of the patient look forward to a rapid cure. Sometimes,—not rarely, I am sorry to say,—the surgeon lacking experience, or, it may be, being too enthusiastic regarding a favorite method, finding all subjective symptoms removed and an apparently arrested deformity, proceeds to remove the support, and to discharge the patient as cured. Following some slight indiscretion, perhaps,—but this is not always necessary—a relapse undeceives both patient and surgeon. These are very unpleasant facts but clinically they need no demonstration. A very considerable experience with the plaster jacket and other methods of support, warrants me in saying that I do not believe any apparatus is capable of accomplishing such uniformly good results as those claimed for the jacket. I have seen many excellent results follow the use of the antero-posterior support long before the introduction of the plaster jacket. A conscientious use of the latter shows inferior results, and the apparatus is open to objections which will be mentioned later.

It is attempted, in using the gypsum bandage in the treatment of Pott's disease, to maintain that position of the spine which is acquired by suspension, by means of the plastic material employed. For the reason that the bandage encircles the thorax and abdomen, it cannot, as a bandage, pass above the axillæ. It becomes obvious, that, as a support, the plaster jacket, *per se*, ceases to be operative above the seventh dorsal vertebra. (Of 666 cases of Pott's disease treated at the Orthopædic Dispensary prior to 1876, 516 were either cervical or dorsal.) However, in order to make the gypsum base operative for disease in the upper dorsal, and cervical regions, a curved iron bar is imbedded, by Dr. Sayre, in the plaster and passed over the head,[1] with which the attempt is made to extend the spine, or to support the head by a submaxillary and an occipital strap. That this method must prove inoperative as an effective means of extension is obvious, for what child could, or would, tolerate an absolute extension force to the head for a length of time sufficient to cure a spinal caries, at, for instance, the second to sixth dorsal, the region most difficult to control in the whole vertebral column? This ungainly and defective apparatus, as a means of support, cannot be compared to the light and comfortable chin piece devised by Dr. Taylor, which makes the treatment of cervical caries one of the pleasures of the orthopædic surgeon. In any event we would discard the plaster above the seventh dorsal, which limits its use to only ten of the vertebræ, and these ten, the five lower dorsal and the five lumbar, are the most easily controlled and supported by any apparatus. As shown by the statistics quoted above, these ten vertebræ are not so likely to be affected as are the other fourteen.

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[1] This "jury-mast" is pictured in Shaw on "Distortion of the Spine," Supplement, 1825, page 100.

I may state, further, my views in general upon the comparative merits of the antero-posterior support and the plaster jacket. The former acts scientifically upon the principle of a lever with the fulcrum at the point of disease. The points of pressure are the pelvis, which forms the basis of support; the transverse processes of the diseased vertebræ and those immediately contiguous to them (the fulcrum), and the anterior, superior wall of the thorax and the axillæ (the resistance). A sufficient *power* is thus maintained, through the medium of the two uprights of the apparatus, to support the spine in the position acquired by recumbency. In my own hands (though it is not perfect), it has accomplished many excellent results in the treatment of Pott's disease, without any of the objections which pertain to the plaster jacket. It can be easily applied and it requires no more training or ability to adjust it intelligently and effectively, than it does to apply a gypsum bandage. A country practitioner, with a village blacksmith (though this is not necessary) could treat any case successfully with a few practical hints, which would be materially strengthened, of course, by a clinical demonstration. The many advantages which the antero-posterior support possesses (especially the modification I have used), will, I hope, induce many to use it who have perhaps, been led to infer from the exaggerated statements of the foremost partisans of the plaster treatment, that the gypsum bandage forms the basis of the only scientific mechanical treatment.

The principal advantages of the antero-posterior support are, 1st. The ease with which it can be adjusted, and the great comfort experienced by the patients who wear it.

2dly. It can be removed with safety at any time by placing the patient in the prone position, when such modifications can be made as are necessary to the comfort of the patient, or the treatment of the case. 3dly. The concentration of the requisite pressure at suitable and convenient points without interfering with transpiration or respiration, and finally, the cleanliness and lightness of the whole apparatus; matters which certainly ought to be consulted in a long and necessarily tedious treatment.

On the other hand, the objections to the plaster jacket are, 1st. Its great weight and the necessary occlusion of so large an area of skin. 2dly. The great danger of excoriations which may develop any time and remain hidden for many days or weeks. 3dly. The absolute necessity of suspension each time the curvature is inspected or the patient cleansed. 4thly. Its great filth, and lastly, its failure to accomplish, in the great majority of cases, for reasons I have assigned, the objects for which it applied. Among those who are able to purchase a steel support, there is certainly no necessity for the adjustment of a plaster jacket, for the simple reason that all that the plaster apparatus accomplishes and more beside, can be accomplished by a suitably adjusted and accurately fitting antero-posterior support; while among the New York City poor, where so many are huddled together in tenement houses, and absolute cleanliness is very difficult of attainment, a plaster jacket soon becomes a nest for all sorts of vermin. Several dispensary patients have begged me to remove the jacket on this account, and more than one, after repeated trials, has declined to have the plaster splint readjusted, because the projection steadily increased under its use.

I have earnestly endeavored to weigh the merits of the plaster jacket as a mechanical aid in the treatment of

Pott's disease. I regret that I can find so little to commend, and so much to condemn, regarding its use. I can safely say that with less trouble, though at a little greater expense, much more satisfactory results can be obtained by the intelligent use of the antero-posterior support. I do not mean to say that the gypsum splint should be wholly discarded in the treatment of Pott's disease. It may be made very useful—where the pecuniary condition of the patient will not permit the expenditure of the small amount necessary to purchase a suitable antero-posterior support—in the comparatively limited number of easily controlled cases occurring below the seventh dorsal. In this region, if applied by the surgeon himself, it affords a much better support than *any* other form of apparatus, adjusted by an instrument maker at the request of the physician, who, through inexperience or indifference, delegates this essential part of the treatment to the uneducated mechanic. It is not the fault, either of the medical profession at large, or the students who are now attending lectures, that they have not been, and are not taught, comprehensively, the subject of mechanical therapeutics as applied to the treatment of chronic and progressive deformities. Dr. Sayre, for instance, places himself on record as saying that he employs the plaster jacket "to the exclusion of all other methods of local treatment"[1] in Pott's disease. Among the other teachers of general or orthopædic surgery in our colleges I have not succeeded in finding one who gives a systematic course of instruction upon the various and complex mechanical indications to be met in the treatment of this insidiously progressive lesion. Instrument makers, who make no pretensions to special training or anatomical knowledge;

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[1] *Spinal Disease and Spinal Curvature.* By L. A. Sayre, M.D. Smith Elder & Co. 1878. Page 1.

mechanics who know nothing whatever of the pathology of the disease, are the source to which many practitioners look for assistance or advice, after leaving college, in treating a case of joint disease. And some of these mechanics, being under no obligation whatever to follow the ideas of the surgeon who first devised a given splint for any purpose, are very prone to introduce so-called "improvements," which, in many instances, render the apparatus practically inoperative. And hence it sometimes occurs that a general practitioner, even if he wishes to use a given form of apparatus, is not certain to obtain the appliance he seeks.

I have no doubt that much of the ill repute that attaches to many forms of apparatus, and many of the failures that have resulted from their use, have arisen from one or both of these causes. And this state of things will continue, and many valuable aids will be lost to the profession, until the student is taught with system and detail, in the various medical colleges, to do with *all* forms of apparatus, which have proved of value in the treatment of deformities, as he has been taught to do with the plaster jacket, viz., to first understand the principle of the application of the apparatus, and then to perform his own work untrammelled by untrained mechanicians. Upon this basis the surgeon will find that the treatment of any case of Pott's disease, with an efficient form of antero-posterior support will prove far more satisfactory to himself, and far more comfortable to his patient, than the gypsum apparatus, which not only necessitates injurious procedures in its application, but which fails, for reasons already pointed out, except in a limited number of cases, to accomplish satisfactorily the object for which it was devised.

Clinical experience has taught me to divide the vertebral column into three regions, so far as the mechanical treatment of spinal caries is concerned. The *first* region includes the lumbar and the last five dorsal; the *second* comprises the first to the seventh dorsal,—both inclusive; and the *third* includes all the vertebræ above the first dorsal. The mechanical treatment of chronic spondylitis in these three regions presents either peculiarities or difficulties which modify not only the treatment but also the prognosis. I feel warranted, therefore, in calling especial attention to them.

When the disease occurs in the first region (the dorsolumbar) the mechanical problem is very simple. It is comparatively easy to adapt any of the various antero-posterior supports in use with the effect of preventing any considerable increase of deformity. With a tolerably firm pelvic base, the axillæ and the antero-superior wall of the thorax (being situated higher up than the diseased point) may be made the means of maintaining the requisite degree of fixation and support. As a rule also, Pott's disease, occurring in this region requires the minimum amount of watching, and the greater number of cases reported, by many writers, as cured, are selected from this region.

In cases involving the middle region (the superior dorsal) we find many difficulties presenting in the way of securing an adequate degree of fixation. The indications are to support a rigid projection in the middle of an, otherwise, flexible column. In addition to this, we have to contend with the constant traumatism which results from the respiratory movements of the ribs. In this region it is very difficult to adapt any apparatus which will satisfactorily meet all the indications. The antero-posterior support, starting with the pelvic vase, sends a long arm to the projection, and its efficient leverage is affected by the fact that the upper arm, held by the superior thoracic and axillary straps, is too short to exert an adequate counter-pressure. If we surmount the antero-posterior uprights with any conceivable head-rest, we cannot, even then, secure the vertebral column satisfactorily. The plaster jacket, in this region, as I have already pointed out, is inoperative, and the ungainly

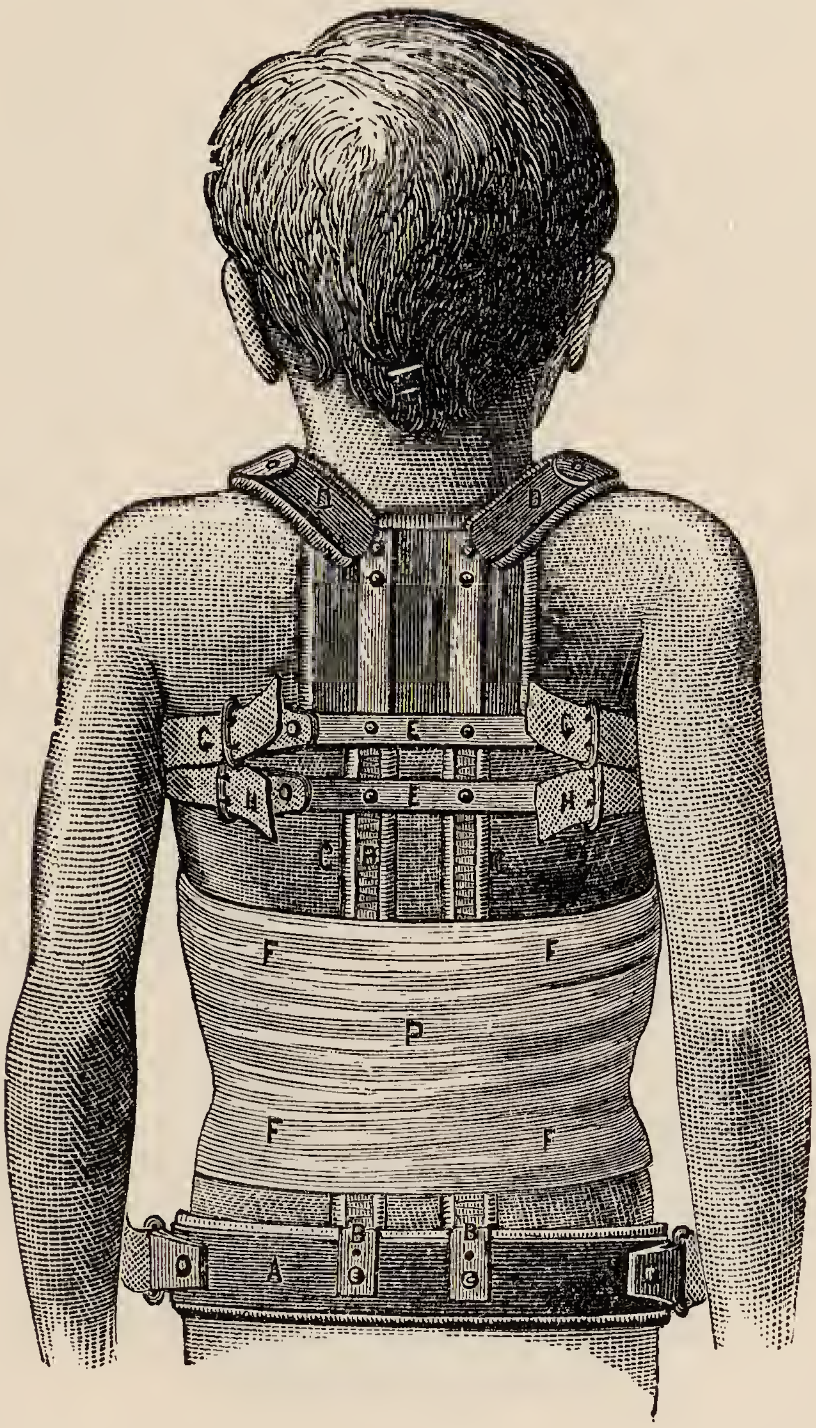

FIG. I

"jury mast,"—which I saw used in the Hospital for the Ruptured and Crippled, as long ago as 1863—exerts no competent force. Taylor's chin piece, acting as a long,

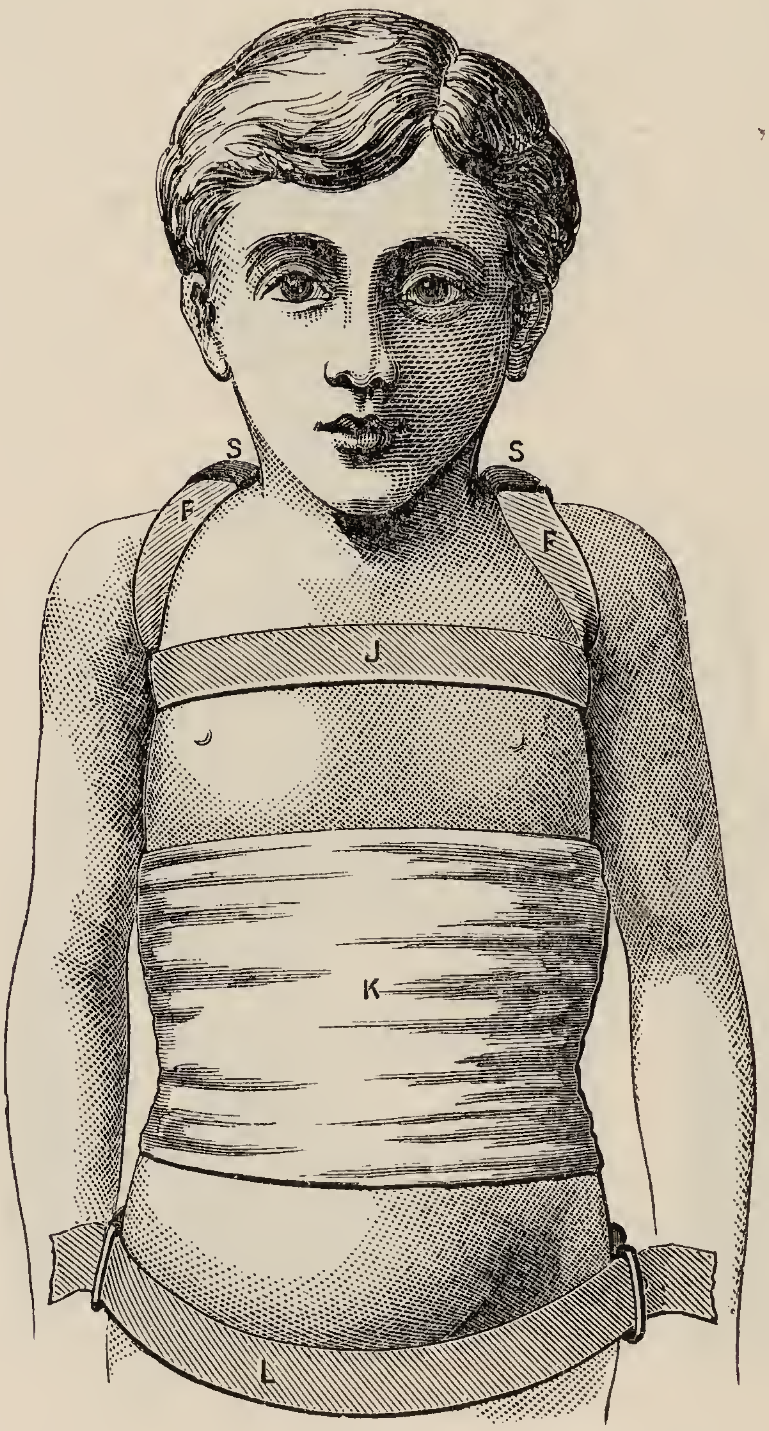

FIG. 2

superior arm of the lever, affords the best medium, in my own experience, of securing efficient support;—not by extending the spine, as some have thought; nor yet by acting as a, strictly speaking, antero-posterior support, as has been advanced by others. Recession of the inferior maxillary occurs if it be used continuously on the latter principle. It has answered best, in my hands, as a simple, but firm and unirritating head rest, limiting the anterior flexion of the head and spine, and removing the greater part of the weight from the vertebral column.

In the third region (the cervical) the mechanical elements again become much easier of adaptation, and some of the best results I have ever witnessed in Pott's disease have been obtained in this region by the use of the chin piece surmounting the two lateral uprights of the antero-posterior support. The objection to the use of a plaster base, simply for the purpose of mounting a "jury mast" upon it, must be evident to any one, when some simple support laced to the body, such as Knight's apparatus, would answer the same purpose and allow also of frequent ablutions. But as it has proved inoperative in my own hands, and as it is not only ugly in appearance, but inflicts unnecessary pain and humiliation upon the patient, I have discarded it.

In the treatment of Pott's disease in the first region, I have found a modification of Taylor's antero-posterior support, secured with a plaster zone, to answer an excellent purpose. This apparatus is pictured in figs. Nos. 1 and 2.

NOTE BY THE AUTHOR

September, 1923

A detailed description of the apparatus is omitted, as well as the histories of several typical cases and the results of treatment. Attention is called to the chin piece (See Figures Nos. 3 and 4) which surmounts the uprights of the spinal brace. The author long ago discarded the "plaster zone." Like all plaster of Paris, spinal supports it is cumbersome and fails to give support a few days after it is applied. In its place he uses a properly fitted belt, covering the affected region,—easily laced to the desired degree of pressure, easily removed for cleanliness, and for the inspection of the pressure lines over the transverse processes.

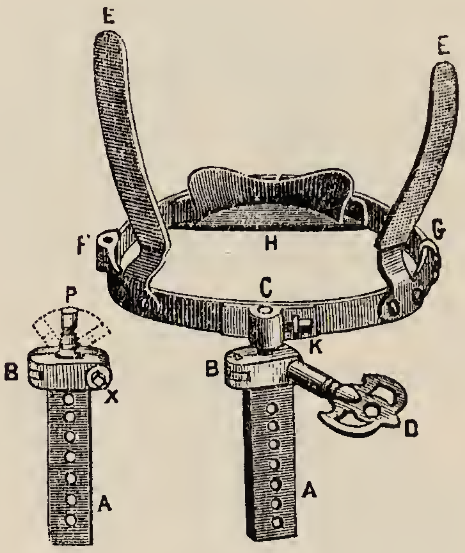

FIG. 3 FIG. 4

Referring to page 66; the author believes that he was the first to record definitely and to specifically describe a case of rotary lateral curvature due to poliomyelitis—(See case of H. D. P.) and he begs to call attention to the remark made regarding the conditions found in rotary scoliosis—"these cases form however a distinct group yet the ultimate muscular condition reminds me of that which exists in the lower extremities after infantile paralysis."

ON THE BENIGN COURSE OF ABCESS IN POTT'S DISEASE UNDER EFFICIENT MECHANICAL TREATMENT.[1]

Revised remarks delivered at the fifth annual meeting of the American Orthopædic Association.

My early medical education was received under the direction of one of the most conservative medical men I have ever met—viz., the late Dr. James Knight, the founder of the New York Society for the Relief of the Ruptured and Crippled. Some of the present members of the American Orthopædic Association also received their first orthopædic training under the same auspices, and with them I can recall the feeling of criticism—not to say ridicule—with which we regarded Dr. Knight's treatment of "cold" abscesses in Pott's disease and in hip-joint disease. Those of our number who passed several years under his preceptorship have had, since we left the institution, ample opportunities to compare his method with the various operative methods; and I think it is a safe thing to say that we are not, even at this date, entirely in accord upon the question of the treatment of chronic tubercular abscess of the spine or major joints.

My own experience has been a varied one. When I left the institution I felt that I had escaped from an unwarrantable restraint, and, with the enthusiasm of my years, I went to the opposite extreme and fell into the error of accepting Sayre's adaptation of the old proverb, "An empty house is better than a bad tenant," a saying, I am assured, that has done much harm as applied to tubercular abscess in chronic articular disease. Experience has taught me that if a qualification could be added to Sayre's dictum, it would express the real state of the case. And this qualification is, "when one can be sure that the tenant will behave well under eviction, or that the tenant will not damage the whole house before he leaves."

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[1] *The New York Medical Journal* for February 20, 1892.

I feel that surgeons generally stand as a unit on the subject of the surgical treatment of *acute* abscess; and if the *chronic* abscess in tubercular disease resembled the acute abscess in its more important particulars, we should not hear the animated discussion that always follows the introduction of the question of the treatment of abscesses arising from tubercular joint disease. And I think it is correct to assume that we are all too apt to regard "abscess" in its generic sense rather than in its pathological sense—that we are too apt to regard a fluctuating tumor, containing the products of a chronic, infectious disease, as a something to be got rid of at almost any cost—ignoring the fact that the contents of a tubercular abscess differ in many important respects from the contents of an abscess due to an acute, non-tubercular lesion. How many of us would hesitate about the propriety of opening a well-marked acute perityphlitic abscess? How many of us would deliberately open a chronic intrapelvic abscess due to an active and progressive tubercular disease of the dorso-lumbar spine?

The conservatism of Dr. Knight amounted practically to a surgical nihilism. The extremists who would open every tubercular abscess connected with an actively diseased spine or joint are, I think, as much in error as those who ignore the indications which point to surgical interference. Unfortunately, with all our discussions on this subject, we have no statistics to demonstrate either the weakness or the strength of either position; but I feel safe in stating that more recoveries occur under the plan of non-interference than under the one which indiscriminately applies the knife to every chronic articular abscess.

My own results—after I adopted the plan of opening every chronic abscess—were not so satisfactory as those which followed non-interference. It is true that at that time the antiseptic method of Lister was not available, and the tubercular bacillus had not been discovered by Koch. After the antiseptic method was introduced I followed it very closely, but still I found my results in chronic abscess of joint disease were not satisfactory. It seemed to me that in opening a tubercular abscess from Pott's disease, for example, we were treating a "symptom" rather than the disease; that we were tapping a reservoir, and paying but little attention to its source; and that we were in too much haste to give exit to the so-called "pus," which kept on flowing from the source, notwithstanding our external antiseptic dressings. It seemed that high temperature would frequently develop, notwithstanding the gauze and protective and irrigation and drainage, and that repair, as a rule, was delayed rather than promoted by our efforts to find a short route to recovery.

After an experience among many cases and many methods, I came gradually to adopt a course which appeared to me to have a rational foundation. I attempted to insure as perfect a mechanical protection as possible to the diseased joint or spine and to maintain the general health in every available way, hoping that the actual disease might cease before the abscess opened (and this proved to be the case on many occasions), or to await the occurrence of either severe local or important general symptoms, due to the abscess itself, before I resorted to incision, etc. After I adopted this plan, I found to my surprise that many abscesses entirely disappeared; that some became quiescent or encysted; that few, if any, gave rise to trouble; that those which opened spontaneously almost uniformly did well; and that my results were more satisfactory and more permanent.

My experience is that tubercular abscesses in Pott's disease, as well as in the abscesses of the chronic tubercular lesions of the major articulations, pursue a very benign course under efficient mechanical protection to the diseased articulation, and that we too often resort to the use of the knife. If the *disease* is cured, then the abscess becomes a *local* affair and we may treat it as such, though it then usually disappears spontaneously.

Among the many cases I could submit to illustrate this position I cite the following:

Master S. W., aged eight years, consulted me on March 4, 1887, bringing a letter of introduction from Dr. Weir Mitchell. The patient had Pott's disease of the spine, involving the eleventh and twelfth dorsal vertebræ. There was a slight kyphosis, which had evidently been progressing for over a year, but which had been unnoticed until it was discovered by Dr. Mitchell. The patient was pale and thin and in a generally bad condition, but gave a good hereditary history. The prominent feature of his case was the presence of three large abscesses—one in the pelvic cavity, another in the gluteal region, and a third on the inner aspect of the thigh—all on the left side. Fluctuation could be detected between the femoral and pelvic abscesses. The gluteal abscess seemed not to be connected with the others. Both the gluteal and femoral abscesses were very large, the former being especially prominent, while the latter increased the circumference of the thigh three inches over the measurement of the thigh of the unaffected side. The pelvic abscess extended nearly to the free border of the ribs. The patient had only a slight rise of temperature, the daily evening temperature averaging 99.6°, the morning temperature averaging 98.2°, for a period of over a month during which the record was kept. The analysis of the urine showed nothing abnormal, and all the vital organs were in good condition.

An antero-posterior support (a modified Taylor's) was carefully adjusted, though at first it seemed difficult, on account of the gluteal abscess, which was so large and extended so high up that it interfered with the hip band of the apparatus. Special provision being made for this, the apparatus was adjusted and the patient was closely watched. He went to his home in May after ten weeks of careful treatment, during which there was a very perceptible decrease in the size of all the abscesses, and in July, 1889, they had wholly disappeared. Careful attention was paid during treatment to the general health and to the climatic surroundings of the patient. He did not spend one day in bed on account of his spinal disease during the entire treatment. I saw the patient during the present summer (1891), and he is well and strong and as active as many boys of his age. As he was so active, I advised that a very light apparatus be worn as a matter of precaution simply, though I have not regarded the patient as being under treatment since May, 1890. The curvature has not increased.

On November 5, 1890, Miss J. B., of Brooklyn, aged ten years, consulted me, bringing a letter of introduction from Dr. Samuel T. Hubbard. The patient had Pott's disease of the first and second lumbar vertebræ, with abscesses very much like those existing in the case just related—viz., large intrapelvic, gluteal, and femoral abscesses—the two last named being especially large and prominent. The patient did not have any rise of temperature above the normal. The antero-posterior spinal apparatus was applied, and the patient was brought to me from Brooklyn every week. Constitutional remedies were used, and the patient was instructed to go out of doors every pleasant day. At the end of two months there was a perceptible decrease in the size of all the abscesses, and at this date (September, 1891) fluctuation can not be detected at any point, and the patient is in remarkably good health. The apparatus is still worn, and the patient is still under professional observation. The kyphosis has not increased.

Both these patients have completely recovered without any increase of deformity. The first named, the boy, is at boarding school, playing football; the second, the girl, is well and going to school, exercising in all respects like her playmates.

I have related the histories of these two cases, among many others that might be cited, because the abscesses were very large; and because they may be regarded as extreme cases. Simple abscesses in Pott's disease do not occasion me any anxiety, and I have not a single case to record in private practice where, after deciding to pursue the policy of non-interference, I have had occasion to regret it. The cases that have been the most troublesome and the most unfortunate are those in which the abscesses have been opened when the indications for so doing were not plainly evident.

This is only the experience of a single individual. I submit it as a contribution to the study of a very important subject.

NOTE BY THE AUTHOR

September, 1923.

S. W. went through his college course without incident. He was prominent in athletics.

I next hear of him as the War Correspondent of the London *Times*, during the Russian-Japanese War, where he served, at the front, under many hardships, during the entire campaign.

Later he appeared as an active participant in the World

War, being engaged in many of the battles on the French frontier, returning with the rank of Major.

I have lately seen him. Somewhat broken in health, as the result of his arduous campaigns, he is again at work. The disease for which I treated him has never given him any trouble.

Miss J. B. recovered entirely and I am informed that she is married, and the mother of children.

ON THE QUESTION OF NON-INTERFERENCE IN ABCESS OF CHRONIC TUBERCULOUS DISEASE OF THE JOINTS.[1]

A Critical Analysis of Thirty-five Cases occurring in the Service of the New York Orthopædic Hospital.

Read before the New York Academy of Medicine, October 17, 1895.

THERE is probably no subject which has come before the Orthopædic Section of this academy, or the American Orthopædic Association, which has occasioned more discussion or a wider difference of opinion than that which involves the treatment of tuberculous abscess of chronic joint disease. In both of these bodies the matter has been widely discussed, and many statements have been made regarding the treatment of abscesses; but there have been no actual statistics presented of the results obtained. On one occasion—the fifth annual reunion of the American Orthopædic Association, held in Washington, D. C., in connection with the Congress of American Physicians and Surgeons, in September, 1891—I ventured to read a paper On the Benign Course of Abscess in Pott's Disease under Efficient Mechanical Treatment.[2] The discussion which followed was interesting and instructive. But the writer found himself, as he has on some other occasions, confronted by a somewhat impulsive and dogmatic expression of opinion. As usual, there was on either side scarcely more than a mere expression of opinion—there were no statistics. It was under these circumstances that the writer promised that he would treat all the patients with tuberculous abscess in the New York Orthopædic Hospital upon the plan of non-interference, using only efficient mechanical treatment and constitutional remedies, and that he would make a formal statistical report of the results; the question being, Which is the better way to treat the ordinary cold abscesses of tubercular joint disease: shall they be freely incised with the usual aseptic precautions, or shall they, under ordinary circumstances, be allowed to follow their own course practically unmolested?

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[1] *The New York Medical Journal* for February 29, 1896.

[2] *Vide New York Medical Journal*, February 20, 1892, and *Transactions of the American Orthopædic Association*, vol. iv., p. 178.

I suppose to many minds this question seems absurd—so strongly is the operative side of the question intrenched in the minds of the general surgeon. But a long experience has brought with it to the writer certain conclusions which differ from the conventional practice of the day, and it is with the hope of throwing some light upon the subject that I stand here to-night to redeem the promise made to my colleagues of the American Orthopædic Association over four years ago.

In order to place the matter fairly before my hearers it seems only right that I should make some extracts from the essay above referred to—that essay being, so to speak, my text for this evening.[1] * * *

I have operated on several patients in similar conditions, and I have seen other surgeons operate in abscesses of this class. The results have been unfortunate. In almost every instance a prolonged chronic septicæmia has followed, with death as the result in the majority of cases, and this is particularly true of adults and adolescents.

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[1] The extracts from the essay on the Benign Course of Abscess, etc., are omitted in this edition. Vide preceding essay.

On the other hand, under the plan of non-interference, with efficient mechanical treatment, I have seen many happy recoveries, of which the two patients reported are only examples, and in the adult and adolescent a favorable prognosis may frequently be given, where incision and drainage would at least be followed by uncertain results. But the *sine qua non* is efficient and prolonged mechanical protection. Unless this can be carried out with almost the same care the surgeon gives to his aseptic dressings the result will be doubtful.

Indeed, the value of mechanical treatment can not be overestimated. It is absolutely essential to success. It does not answer to simply apply a support and then leave the patient to the care of the instrument-maker. The surgeon must feel the same responsibility regarding the attention to details that he does in treating a fracture or in following up the dressings after a major operation. He must know that the mechanical treatment is doing the work required of it and that real support is being secured.

The method also is important. For example, recumbency in Pott's disease, except as a temporary expedient, is a fallacy; so is the weight and pulley in hip joint disease. The various portable frames and beds, the leather and felt jackets do not afford proper protection to the diseased conditions under consideration. The fundamental principle of mechanical treatment in chronic tuberculous joint disease is: *Protection to the diseased part, with the maintenance of functional activity of the other parts of the body.* The plaster-of-Paris jacket is not adapted to the treatment of Pott's disease, and I should no more think of applying it to the treatment of this condition than I should of opening an abscess without aseptic precautions. Long ago, in 1879, in my brochure on Pott's disease, I called attention to the fact that the gypsum splint was useless in chronic spondylitis above the eighth dorsal vertebra. When the disease is below that point we have a far more accurate means of support than the plaster-of-Paris jacket, and a well-adjusted ball-and-socket chin piece is so far superior to the "jury mast" as a support above the eighth dorsal vertebra that the latter should be relegated to the eighteenth-century practice where it belongs.

With an accurately fitting antero-posterior steel support, based upon the Taylor principle, supplemented when practicable by a properly applied belt, making pressure upon and limiting motion at the diseased part of the spine, we have as nearly as may be an ideal protection for the diseased spine.

Why should we limit superior thoracic respiration in dorso lumbar Pott's disease, or why should we interfere with abdominal respiration in cervico-dorsal disease? The ideal apparatus should support the diseased portion of the spinal column and allow, so far as practicable, functional activity at the non-diseased portion. This certainly can not be accomplished by any encasing jacket which attempts, though it does not succeed, in satisfactorily splinting any part of the spinal column.

What is abscess? Technically speaking, it is "a collection of pus formed as the result of suppuration."[1] For the purpose of this paper we must call a collection of tuberculous material an abscess, though it frequently does not contain any pus at all. If the abscess of tuberculous joint disease was the result of a pyogenic process this paper would probably not have been written, and there probably would be no important difference of opinion regarding the question of the treatment of abscesses among surgeons. But the cold abscess of tuberculous diseases presents, both clinically and pathologically, an entirely different aspect from

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[1] Foster's *Encyclopædic Medical Dictionary.*

"a collection of pus, formed as the result of suppuration." Clinically it presents a simple fluctuating tumor. There is no apparent heat, and the general temperature is normal, or nearly so. Pathologically it is not a pyogenic abscess. It contains the product of a distinct condition having the bacillus of tuberculosis as its cause. Ogston, Cheyne, Collins Warren, Senn, and latterly Dr. John Dane, of the Children's Hospital in Boston, as well as others, have proved that these abscesses are sometimes absolutely sterile. Why then should we be in a hurry to incise the abscess and permit the entrance of micro-organisms?

Ordinarily the tuberculous abscess pursues a benign course. Frequently we are obliged to search for it, especially in dorso-lumbar Pott's disease. The patient may be wholly unaware of its existence. It gives rise to no inconvenience. It is simply a sign of disease, the location of which is remote from the indolent and ofttimes painless expression of it.

So long as we know of the existence of an abscess in chronic joint disease by our sight or by palpation only, we are perfectly justified in letting it alone. There are no indications for active interference. As has been mentioned, it may be sterile. Then, again, it may be absorbed, or we may cure the *cause* of the abscess before the latter becomes at all pronounced, in which case the abscess becomes a local affair and has no special significance.

But even under the most favorable circumstances the abscess may assume a different phase. The simple tuberculous material may undergo a change and a pyogenic element is added. The so-called "mixed infection" then occurs. Under these circumstances we may find a slight daily rise of temperature, showing a minor degree of septicæmia, and sooner or later the abscess may increase in size and the process of burrowing may begin.

It is in conditions of this kind that the judgment, experience, and patience of the surgeon will be tested. The temptation is to use the knife at once. My advice is, Do not be in a hurry to do so. Wait for a while. The septicæmic flurry may and probably will soon pass over—if you protect the articulation. And if the infection proves to be a slight one, the general condition remaining good, with approximately good appetite, sleep, and digestion—wait. Get your patient up—in his apparatus. Let the healthy parts of the body move in the air and sunshine. Even if the temperature goes to 101° or even 102°, do not be worried. In children this is not a serious menace. And if you watch and wait, and maintain the joint protection, you will probably find that the condition will gradually subside.

But if, as sometimes happens, the infection is very severe and the symptoms become urgent, there is no question as to the course to be pursued. Make a free incision and use ample and perfect aseptic means to protect the abscess. But if you can maintain a secure protection to the diseased articulation it is better by all means, if the infection is slight, to let the abscess alone. And if it must open, let it open itself.

A temperature of 101° to 102° or more in chronic tuberculous disease tells a far different story from the same temperature in acute pyogenic abscess. I have stood many times at the bedside of a patient with tuberculous disease and abscess, watching the vital signs with apprehension, but waiting for some decided sign to guide me, and as I have waited Nature has solved the problem by either a spontaneous opening of the abscess or a spontaneous absorption. And the patient has been a gainer by my waiting.

To those who watch and wait the problem is solved.

Those who watch and wait will, I think, reach the conclusion that I have reached long ago—viz., that the ultimate recovery of the patient with a useful joint is better under the plan of non-interference than it is under that procedure which makes no practical distinction between a tuberculous abscess and a pyogenic abscess, but opens either indiscriminately.

One hears a good deal about the burrowing of abscesses, of the "maceration" of healthy parts by the contained matter, etc. But I have learned, under ordinary circumstances in chronic tuberculous abscess, to have no fear of burrowing, and Nature so far encapsulates a chronic abscess that "maceration" exists largely in the imagination of those who fear it. Surely, in the cases I have reported to the American Orthopædic Association there was ample chance for "maceration" after the extensive "burrowing" that existed. In one case (S. W.) the abscess extended from the diaphragm to the lower third of the thigh, and involved the gluteal region as well. Nearly the same condition existed in the other one (J. B.). But, under efficient treatment, both abscesses were absorbed, and there is not a trace of their existence to-day. So it is in many other instances. The slow burrowing of a chronic abscess need not be feared. The burrowing of an acute pyogenic process demands immediate attention.

Looking at the question in the abstract, we may ask, Do asepsis, incision, drainage, irrigation, etc., meet the indications in our present state of knowledge? Does the knife, in making an external wound which communicates with the diseased joint, favor the ultimate cure of the disease? How often can we be sure, short of a radical excision of the joint (and this is impracticable at the hip and the spine), that we can remove the tuberculous material? Logically speaking, one should go on and excise the joint if one opens a tubercular abscess connected with it; for unless all the tubercular material is removed there is increased danger with a freshly incised wound, and it is impossible to insure perfect sterilization of an open sinus for a prolonged period of time.

There are questions which I have been studying for years. Through all the phases of Listerism, through all the aspects of a moderately active operative work, through the dreary field of non-interference, I have plodded in my desire to seek the truth. I bring these questions before this audience to-night and ask them to aid in the solution of a most important problem.

As a contribution to the subject I submit the results of four years of patient work in the Orthopædic Hospital. I have no other statistics with which to compare them; but I sincerely hope that some of my colleagues who hold different views will give their side of the question in an equally practical form, so that we may see both sides of the question.

From the first day of May, 1892, to the present date, sixty-three patients with hip disease, eighteen patients with Pott's disease, and twelve patients with knee-joint disease (ninety-three in all) have been treated in the New York Orthopædic Hospital.

During this period Dr. P. H. Fitzhugh and Dr. R. A. Hibbs have respectively filled the position of house surgeon, the former serving to the 1st of January, 1894, the latter being the present incumbent. Their careful work in watching the patients under their care and their thoroughness in keeping the histories, etc., deserve my warmest commendation.

Of these ninety-three patients with joint disease thirty-five have had abscesses. Nineteen of them occurred in the service of Dr. Fitzhugh and sixteen in the service of Dr. Hibbs.

Only thirteen of Dr. Fitzhugh's cases and the same number of Dr. Hibbs's cases come accurately within the scope of this report, which is to demonstrate that, in the great majority of instances, tuberculous abscess in chronic joint disease pursues a benign course under efficient mechanical protection, and that most excellent results are obtained under the plan of non-interference with the abscess. Hence, it would be fair to report those cases only which have been watched from beginning to ending, and where the treatment has been carefully and continuously carried out. The last six of Dr. Fitzhugh's cases are not by any means representative ones, nor are Cases XXIII, XXXIV, and XXXV of Dr. Hibbs's series; but I feel that it is better to report *all* the abscesses that have occurred in the hospital, so that the hearer may judge for himself and draw his own conclusions.

The treatment pursued in these cases was:

1. Efficient and continuous mechanical protection (*a*) with the long Taylor traction splint in hip-joint disease; (*b*) with the antero-posterior spinal support in Pott's disease; and (*c*) with an immobilizing or traction apparatus in knee-joint disease.

2. By constitutional remedies—cod-liver oil, the hypophosphites, malt, iron, etc.

3. By rest in bed during the treatment of the deformity stage of hip-joint disease and recumbency in the acute phases of Pott's disease. The patients, however, were up and about nearly all the time, the hip deformity yielding easily under ordinary circumstances to the hip-splint traction upon an inclined plane.

4. In all cases the abscess was allowed to open spontaneously, no pressure or force being used, aseptic dressings were applied at once just as if an artificial opening had been made. In most instances the simplest external dress-

ings were used. In a few cases irrigation and drainage were employed. In the majority of instances all that was done was to keep the parts clean externally by the use of peroxide of hydrogen, bichloride-of-mercury solution, carbolic-acid solution, europhene, etc. All temperatures were taken at the rectum. No iodoform was used.

The following are the condensed histories, photographs of the result being presented in all available cases.

Dr. Fitzhugh's cases are as follows:

Case I.—Tommy L., aged four years; dorso-lumbar Pott's disease; large abscess, involving pelvic cavity and thigh; duration of disease before abscess appeared, eight months; abscess opened spontaneously one month after its recognition; closed four months later. Treatment: Poultice before abscess opened; after, simple external dressings; no irrigation, no washing out. There was no constitutional disturbance and no increase in deformity. Patient discharged cured.

Case II.—George R., aged twelve years; hip disease; ilio-femoral abscess primarily, afterward involving outer aspect of thigh; abscess very large; duration of disease before abscess appeared, five months; abscess had entirely disappeared one year later; no constitutional disturbance; no local treatment; no subsequent recurrence of the disease; recovery complete with half an inch shortening; joint motions almost normal; has been working in a store over two years.

Case. III.—Ella G., aged six years; double hip-joint disease; abscess large, ilio-femoral, on left side; no abscess on right side; right hip has completely recovered.

The disease on left side was not recognized until the abscess appeared; it opened spontaneously three months later; a small sinus still remains, discharging a few drops daily; no pain. Temperature during active period of abscess and just after its opening, 99° to 101°; simple external dressings; no irrigation or washing out; general condition excellent; left leg a quarter of an inch shorter than right.

Case IV.—Lewis L., aged two years and a quarter; abscess after excision of hip joint; excision performed in Scranton, Pa., in March, 1889; the abscess appeared at outer aspect of thigh two years after operation; opened spontaneously a month after it appeared; closed five months later; simple external dressings; no constitutional disturbance; recovery; flail joint (due to the excision); three inches shortening.

Case V.—Minnie G., aged five years; hip-joint disease; had two abscesses—one on the anterior aspect of the thigh; the other, in the gluteal region, was quite large; the thigh abscess occurred thirteen months after the first appearance of the disease; the gluteal abscess two years and four months after first appearance of disease. Thigh abscess opened spontaneously nine months after it was first noticed; the gluteal, one month; thigh abscess closed in eight months; gluteal in four months. Treatment, simple external dressings. Temperature after gluteal abscess opened for two weeks, 99° to 104°; since then, normal. Present condition: Extension of thigh to a hundred and seventy-five degrees, with ten degrees of motion; no lateral deformity; abscesses have remained closed; shortening, a quarter of an inch.

Case VI.—Nettie D., aged two years; hip disease; gluteal abscess of about the size of an orange; duration of disease before abscess appeared, three years and one month; abscess has entirely absorbed after existing for one year; general condition most excellent. Present condition: Extension to a hundred and seventy-five degrees; about ten degrees of motion; slight adduction; one inch shortening.

Case VII.—Eddie P., aged eight years; hip disease; anterior femoral abscess, size of a goose egg; disease existed eighteen months before abscess appeared; opened spontaneously three months later; a small sinus still discharges a drop or two daily. Temperature, 99° to 101° for some months, but there was no interference with appetite, sleep, or exercise. Present condition: Extension to a hundred and sixty degrees; ten to fifteen degrees of motion; very slight adduction; shortening, a quarter of an inch.

Case VIII.—Joseph R., aged eight years; Pott's disease, dorso-lumbar; large psoas abscess, filling one half of pelvic fossa, existed on admission; eighteen months later, abscess had entirely disappeared; no constitutional symptoms; no increase in deformity.

Case IX.—Elizabeth O., aged seven years; abscess after excision of hip joint; small abscess appearing at line of cicatrix; excision performed at a general hospital in this city; abscess appeared in March, 1893, opened spontaneously one month later; closed in three months; simple external dressings; no elevation of temperature; an inch shortening; walks fairly well with telescoping joint, due to the excision.

Case X.—Frank O'N., aged seven years; knee-joint disease; large abscess over internal condyle and involving popliteal region; abscess existed at time of admission; increased very rapidly and opened spontaneously four months later; about six weeks after abscess opened patient left the hospital and went to Buffalo. At that time the sinus was in excellent condition, discharging a few drops only every twenty-four hours; no constitutional disturbance; no irrigation; simple external dressings.[1]

Case XI.—Alice D., aged two years and a half; hip-joint disease; very large anterior femoral abscess, involving also outer aspect of the thigh; disease existed two years before abscess appeared; fifteen months later abscess has entirely disappeared by absorption without the slightest constitutional symptoms; hip was exquisitely sensitive when patient entered hospital; strongly marked tubercular heredity. Present condition: Extension to a hundred and eighty degrees; flexion to a hundred and forty degrees; no lateral deformity; shortening, half an inch.

Case XII.—George S., aged ten years; hip-joint disease; large gluteo-femoral abscess, which was present when patient entered ward, having been present ten months prior to that date; five months after admission abscess opened spontaneously; had four sinuses, three of which have closed; one is now discharging a few drops daily; nearly healed[1]; no constitutional disturbance; general health excellent; simple external dressings; shortening, half an inch.

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[1] The patient went under the care of Dr. Bernard Bartow, of Buffalo, who reports to Dr. Fitzhugh that the sinus has closed.

CASE XIII.—James S., aged five years; Pott's disease, dorso-lumbar. On admission, iliac abscess, with small sinus discharging. In very bad condition on admission; thigh flexed to ninety degrees; sinus closed two months after admission. Temperature at first, 99° to 102°. This soon disappeared and patient made rapid improvement; simple external dressing; no increase in deformity; patient well.

CASE XIV.—Robert K., aged ten years; Pott's disease, (1) upper dorsal, (2) dorso-lumbar; two distinct foci of disease; large abscess in left iliac fossa; abscess increased in size for a time; general condition very much improved; no constitutional disturbance; no increase in deformity; patient removed by parents after a nine months' residence in hospital in October, 1893; was last seen May 23, 1894. Prior to that date abscess had been aspirated, and it subsequently opened spontaneously in February, 1894. A small sinus was discharging when last seen. Patient was in good condition and going to school.

CASE XV.—Yvonne O., aged five years; Pott's disease, dorso-lumbar; large abscess in right iliac fossa; opened spontaneously twelve months after admission; very profuse discharge; irrigation, etc., for several weeks, during which temperature ranged from 99° to 103°. Afterward simple external dressings were used. After being in the hospital for a year was removed by her mother (July, 1892) to the country, the sinus still discharging; spinal deformity slightly increased. She also had a fluctuating tumor involving the upper third of thigh (left). Patient has not been examined since last March (1892), when it was reported that she was in good condition, able to go about, with several sinuses discharging.

CASE XVI.—Nellie C., aged ten years; hip disease; small femoral abscess, which opened spontaneously two months after admission. Irrigation and antiseptic dressings. Temperature before abscess opened, 100° to 101°;

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[1] *January 15, 1896.*—All the sinuses have closed.

after opening, 100° to 104°. A second abscess appeared about a year later on inside of thigh, which opened in a month. Irrigation, etc., for a short time, afterward simple external dressings.

The patient was doing well and improving when she was removed in the summer of 1892 to a general hospital, and after a brief stay she was transferred to another general hospital. She then had five discharging sinuses. She died of pulmonary tuberculosis some time in the winter of 1893.

During her residence in the two general hospitals referred to the patient had little or no protection to the diseased joint. She left the Orthopædic Hospital in a fair condition, walking about on her traction splint. The loss of efficient mechanical treatment was, I believe, an important factor in her subsequent career.

Case XVII.—Morris M., aged thirteen years; Pott's disease, dorso-lumbar. On admission to the hospital in April, 1893, had four discharging sinuses, with marked double psoas contraction. General condition on admission very poor. Improved greatly in general health for a time. Sinuses dressed externally, with occasional irrigation; no marked elevation of temperature. Was discharged from hospital as incurable, after nineteen months, in November, 1894, with ten discharging sinuses. Died in June, 1895, eighteen months later, with waxy liver and general tuberculosis.

Case XVIII.—Mary C., aged ten years; hip disease; admitted to hospital, May, 1893, with small sinus on external aspect of middle third of thigh, which closed one week afterward and has remained closed; no elevation of temperature; simple external dressings; joint can be fully extended, and has about ten degrees of motion; no lateral deformity; general condition excellent; shortening, two inches.

Case XIX.—Maggie G., aged four years; Pott's disease, dorso-lumbar. When admitted to the hospital, in June, 1892, was in a deplorable condition. She had pulmonary tuberculosis, and three sinuses were discharging profusely—one just above the kyphos, the second at the inner aspect of the thigh, and the third just below the trochanter major. Temperature, 101° to 104°. The patient had been treated prior to admission in a general hospital for two years with a plaster-of-Paris jacket, the one we removed weighing almost as much as the emaciated patient. The condition was hopeless from the first, and after two months' effort the patient was discharged. She died shortly afterward of phthisis pulmonalis.

The following cases occurred in the service of Dr. R. A. Hibbs:

Case XX.—Arthur K., aged seven years; hip disease; large abscess on outer aspect of the thigh; duration of disease before abscess appeared, five years; abscess opened spontaneously eight months after its appearance; closed three months later; has been closed one year. Treatment, simple external dressings. Temperature, 99° to 100°. Present condition: Extension of thigh to one hundred and eighty degrees; twenty-five degrees of motion; no lateral deformity; general condition excellent.

Case XXI.—Jennie G., aged seven years; Pott's disease, dorso-lumbar; abscess in either iliac fossa; very large on right side; somewhat smaller on left; abscess appeared about five years after the disease was first noticed. The right abscess disappeared by absorption about eighteen months after it was first noticed; the left also disappeared in about one year. Temperature, 98.4° to 100°. General condition excellent; deformity has not increased.

Case XXII.—James E., aged thirteen years; hip-joint disease. Previous to admission patient had some extensive operation, probably exploratory, at antero-lateral aspect of thigh, the cicatrix presenting a sinus on admission which discharged slightly for a short time. Subsequently, about one year later, an abscess appeared in same location. It gradually became quite large, and opened spontaneously one month after it was noticed. In four months it had healed; no constitutional disturbance; simple external dressings; extension of thigh to a hundred and eighty degrees; flexion to ninety degrees; no lateral deformity; shortening an inch and a half; general condition excellent.

Case XXIII.—Sophie C., aged four years; hip-joint disease; very large gluteal abscess. Disease existed three years before abscess appeared—opened spontaneously ten months later. While under treatment patient developed knee-joint disease on the same side. The gluteal abscess interfered with the perineal pads of the hip splint, and the weight and pulley were used. It was impossible to apply efficient mechanical treatment; abscess continued to discharge, and one day the mother arbitrarily removed patient from the hospital, after which treatment was refused. Temperature rose to 104° after abscess opened for a short time, after which it became about normal. Shortening, one inch. Patient left hospital with considerable flexion and adduction.

This case is especially interesting as showing how the abscess itself may interfere with efficient mechanical treatment.

Case XXIV.—Elmer G., aged eight years and a half; hip-joint disease; anterior femoral abscess of an average size. Abscess was present when patient entered ward. It opened spontaneously soon after we first saw it; closed in twenty-one months. Temperature immediately after opening, 103° to 104°; for a few days after that, 100° to 101°; simple dressings only; abscess reopened seven months after it closed; slight discharge for three months, giving no inconvenience. Present condition: Extension of thigh to a hundred and seventy degrees, twenty degrees of flexion; no lateral deformity; general condition excellent throughout; one inch shortening.

Case XXV.—Mary Z., aged thirteen years; hip disease; extensive abscess on antero-lateral aspect of thigh. Duration of disease before abscess was noticed, seven months; opened spontaneously four months later; entirely closed in eighteen months; simple external dressings; temperature, 99° to 100°; patient showed steady improvement while under treatment; is now in excellent condition; thigh extension, a hundred and eighty degrees;

twenty degrees of motion; no lateral deformity; shortening, an inch and a quarter.

Case XXVI.—Julia H., aged eight years; hip disease, large abscess on outer aspect of thigh. Disease existed twenty-two months before abscess appeared. Opened spontaneously five months later; closed in six months; temperature, 99° to 100°, with occasional rises to 102°; simple external dressings. General condition kept improving during treatment. Present condition: Extension of thigh to a hundred and eighty degrees; twenty-five degrees of motion. No lateral deformity; shortening, a quarter of an inch.

Case XXVII.—Ada M., aged eleven years; Pott's disease, dorso-lumbar; large ilio-femoral abscess on either side. On the right side abscess extended from diaphragm to middle of thigh, both anteriorly and posteriorly. Aspirated twice without diminution of the pus; a slight discharge appeared at the point of aspiration for a short time. This closed leaving a scar with the abscess as large as ever. The whole mass underwent absorption. No evidence of abscess exists to-day. On the left side the abscess was also very large, but did not extend very far below Poupart's ligament. This abscess has disappeared by absorption entirely in about three years. Neither of these abscesses gave the patient any constitutional symptoms. At one time her temperature rose slightly at irregular periods, and, as she came from a malarial region in New Jersey, Dr. Andrew H. Smith thought it was due to this cause. She was up and about all the time in her apparatus. No increase in the deformity. Patient in excellent condition.

Case XXVIII.—Martha H., aged five years; hip disease; large abscess on outer aspect of thigh. Disease existed a year and four months before abscess appeared. Opened spontaneously four months later. Closed in eleven months. Temperature, 99° to 100°, exceptionally to 101°. Simple external dressings. General condition excellent at all times. Extension of thigh to one hundred and eighty degrees; twenty degrees of motion. No lateral deformity. Shortening, a quarter of an inch.

Case XXIX.—Joe B., aged nine years; hip disease; large abscess on lateral aspect of thigh, which existed when patient entered ward. Opened spontaneously six months later. Closed in ten months. Temperature, 99° to 101°. For four months after opening, irrigation with bichloride of mercury or peroxide of hydrogen was used, but the discharge continuing very profuse, this was stopped. After irrigation was stopped and simple external dressings were used, the temperature fell from half a degree to a degree and a fifth every day, and discharge was much diminished. General condition excellent. Extension of thigh to one hundred and seventy degrees; twenty degrees of motion. No lateral deformity. Shortening, an inch and an eighth.

Case XXX.—Samuel L., aged twelve years; hip disease; very large abscess on anterior aspect of thigh, extending down to three inches above the knee joint. The disease had existed two years and three months before abscess appeared. Abscess opened five months after it was noticed. It is now closed, eleven months after it opened. One or two crusts appear on the long cicatrix. Immediately after the spontaneous opening the temperature rose to 104°, and remained about that point for a few days. For four months the large cavity was irrigated twice daily, but the temperature remaining high, about 102° to 103°, and the discharge continuing profuse, irrigation, etc., was stopped and simple external dressings were employed. The temperature at once dropped from one degree to one degree and a half, and the discharge diminished. Patient is now in good general condition. Extension of thigh to one hundred and seventy degrees; fifteen degrees of motion. Very slight lateral deformity. No shortening. A second abscess (gluteal) has recently developed. It has opened spontaneously during the past few days.

Case XXXI.—Abe L., five years old; hip disease; abscess discharging profusely when patient entered hospital. His general condition was very poor. Under simple external dressings his general condition improved very much, and in a year the sinus had entirely closed. Temperature, 98.4° to 100°, with occasional rises to 102°. Present condition: Extension of thigh to one hundred and seventy-five degrees; motion slight—about five degrees; slight abduction; shortening, half an inch.

Case XXXII.—Lulu R., aged seven years; hip-joint disease; abscess discharging when patient entered hospital. Very slight discharge for a year. Sinus closed. General condition excellent. Temperature, 99° to 100°. Present condition: Extension of thigh to one hundred and seventy degrees; ten degrees of motion; slight abduction; an inch shortening.

Case XXXIII.—Samuel K., aged five years; knee-joint disease; small abscess over internal condyle. Disease existed ten months before abscess opened. Abscess opened spontaneously seven months later; still discharging slightly.[1] A second abscess appeared on the opposite side six months ago; opened two months later; closed a few weeks ago. When patient came under treatment the joint was very flail-like. Patient has improved very markedly. Flail-like motion has become much modified. General condition excellent. Temperature, 99° to 100°. Knee joint straight. Motion through eight or ten degrees. No subluxation.

Case XXXIV.—George K., aged twelve years; multiple joint disease. Patient entered hospital in June, 1893, from an almshouse in St. Lawrence County, New York, where he had been for several years. On admission he had disease in both hip joints, left knee, left shoulder, and right elbow. Also extensive necrosis of left tibia. He had in all thirty cicatrices upon admission.

Dr. McBurney, in consultation, advised amputation at left thigh, and the operation was performed by Dr. McBurney at the Roosevelt Hospital. The history is a long one, and to summarize I may say that the disease in the left hip recovered some months after amputation, and he was fitted with an artificial leg. The patient walked well with the hip splint on the right leg and crutches. Disease in right hip recovered. The sinus at the right knee closed. Patient could walk without crutches and with artificial leg. He suddenly developed albuminuria and died of chronic nephritis on September 28, 1895. Post-mortem showed recovery at right hip, with an abscess posterior to that joint, connecting with a diseased bone near the ischiatic notch; other joints were not examined.

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[1] Sinus has completely closed, January 1, 1896.

Case XXXV.—Annie Z., aged eight years; hip-joint disease and lumbar Pott's disease; disease existed three years before abscess, connected with the lumbar disease, opened; it opened seven months after its appearance and discharged profusely; patient developed a cough, and Dr. Chappell, the attending physician to the hospital, and Dr. Andrew H. Smith, consulting physician, diagnosticated pulmonary tuberculosis; temperature before abscess opened, 99° to 102°; after opening, 100° to 104°; irrigation and injections were used, but the patient failed rapidly and died of phthisis in February, 1895.

Summary.—Of the thirty-five patients (all the abscess cases which have occurred in the hospital for over four years), twenty-six remained under the care of the institution for a sufficient length of time to test the value of the plan of non-interference.

Of these twenty-six patients, three had each two distinct abscesses, making twenty-nine abscesses treated in all. In two of the double-abscess cases there were large bilateral ilio-psoas abscesses, and it is worthy of note, especially in connection with the cases of S. W. and J. B., that absorption of the abscesses occurred in all these cases.

Of the twenty-nine abscesses, eight (27.58 per cent.) underwent complete absorption; nineteen (65.51 per cent.), after opening spontaneously, closed under simple external dressings in periods ranging from two to twenty-one months; and in two (6.89 per cent.) there are still small sinuses discharging a few drops daily.

Of the twenty-nine abscesses, 93.09 per cent. have either closed or been absorbed.

Of the remaining nine patients, one was removed by her mother after our efforts, up to the time of removal, had failed to produce an adequate joint protection on account of the location of the abscess. In one instance the abscess was nearly well when the patient entered the wards.

In seven instances the patients either entered the wards with phthisis pulmonalis, or had multiple joint disease, or were removed from the care of the hospital while under active treatment. Of these seven, five died and two have small sinuses which discharge slightly.

In conclusion, I desire to say two things: 1. None of these patients were selected, and none were declined, on account of their condition at the time of their application. 2. I hope that others who hold different views, and especially those who practise incision, etc., will make reports of entire unselected groups of patients with tuberculous abscess of the joints. We shall then have a basis for intelligent comparison.

A CONSIDERATION OF THE PARAPLEGIA OF POTT'S DISEASE WITH ESPECIAL REFERENCE TO THE RESULTS OBTAINED BY MECHANICAL TREATMENT (WITH REMARKS ON THE EFFECT OF DORSO-LUMBAR DISEASE UPON MENSTRUATION.)[1]

An Analysis of Forty Cases Occurring in the Service of the New York Orthopædic Dispensary and Hospital.

When your President kindly invited me a few weeks ago to participate in the discussion of the subject which brings us together to-night, and the part was assigned me of speaking upon the Results of Mechanical Treatment, I found, after making the effort, that the time was too short to make a comprehensive digest of a large number of cases. Dr. T. Halsted Myers had, however, already made a brief and concise contribution to the subject,[2] in which he says regarding the 1,570 cases which he investigated that it was "A number far too large to tabulate." It seemed to me, under these circumstances, that a critical analysis of the available cases occurring in the practice of the New York Orthopædic Dispensary and Hospital and a somewhat rigid scrutiny of the results obtained, would be of service, and with this end in view, and with the kind and most persevering assistance of Dr. P. H. Fitzhugh, of the dispensary staff, I have been able to collect a sufficient number of cases to aid us, I think, in our deliberations.

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[1] *The Journal of Nervous and Mental Diseases*, May, 1897.

[2] "The Prognosis of Pressure Paralysis," *The Times and Register*, Nov. 29, 1890.

In approaching the subject I instructed Dr. Fitzhugh:

1st. To report all the cases available occurring in the service of the institution which had been under its continuous care and which had afforded enough co-operation at home to make the treatment reasonably effective.

2d. All such cases, "good, bad or indifferent," were to be tabulated and reported in full.

3rd. Under no circumstances were the cases to be selected and none, however bad the result, were to be rejected.

The number of cases which we are able to report, in the limited time at our disposal and under these conditions, is forty. With a much longer time for preparation many other cases might be traced, a final examination made, and the result recorded. But it is fair to assume that the cases here reported represent the average results obtained by the methods pursued in the institution in its outdoor practice.

And it seems only right to say in connection with these cases that they all occurred among the poorer classes of the tenement house population; that all of them were treated at their homes under not very favorable conditions, and that they were visited at regular intervals by some member of the dispensary staff, the mother, or some more or less interested relative or friend caring for the patient between the professional visits. Under the conditions which exist in private practice the results obtained are doubtless much better.

All these patients were subjected, generally speaking, to the same treatment. That is, a Taylor spinal brace was applied, with or without the ball and socket chin piece attachment, and necessarily all were treated in the recumbent position—the patient lying on his back upon a tolerably firm mattress, without any pillow. The apparatus was adjusted to that position which would steady and support the entire spinal column without producing undue pressure at any point. After the final adjustment of the apparatus, which took perhaps several days, periodical visits were made, the pressure line of the support was inspected and the apparatus was modified if necessary. Iodide of potassium was not used and no special internal medication was employed—the main principle of treatment being to produce a practical immobilization of the spine—especially of the diseased region.

Several tables have been prepared showing the age, first symptoms, duration and kind of paralysis, location, abscess, results, etc., to which I beg to call your attention before making any extended comment on the lessons they convey.

Twelve tables in all are submitted, to which I beg to direct to your attention.

TABLE No. I.

Age of Patients on admission to the Dispensary.

From two to three years	3 Cases
From three to four years	6 Cases
From four to five years	3 Cases
From five to six years	5 Cases
From six to seven years	4 Cases
From seven to eight years	5 Cases
From eight to nine years	5 Cases
From nine to ten years	1 Case
From twelve to thirteen years	1 Case
From thirteen to fourteen years	1 Case
From fifteen to sixteen years	1 Case
Seventeen years	1 Case
Eighteen years	1 Case
Twenty years	1 Case
Twenty-nine years	1 Case
Forty-three years	1 Case
Total	40

It will be seen that all ages are represented in this table, thirty-two being under ten years of age, seven being above that age, the oldest being forty-three.

TABLE No. II.

Showing the Location of the Disease.

(With a single focus of disease.)

In the cervical vertebræ	2
In the cervico-dorsal vertebræ	3
Dorsal, upper vertebræ	11
Dorsal, middle vertebræ	14
Dorsal, lower vertebræ	4
Dorso-lumbar vertebræ	3
	37

(With double foci.)

Upper dorsal and lumbar	1
Cervico-dorsal and dorso-lumbar	1
Upper dorsal and dorso-lumbar	1
Total	40

Of these forty cases thirty-three were affected in the middle dorsal, upper dorsal or cervical regions. Three had disease both in the dorsal and lumbar regions.

TABLE No. III.

Duration of disease before the symptoms of paralysis occurred.

The disease had existed

Under one year	in	8 Cases
Between one and two years	in	13 Cases
Between two and three years	in	4 Cases
Between three and four years	in	5 Cases
Between five and six years	in	3 Cases
Between six and seven years	in	1 Case
Between seven and eight years	in	2 Cases

Ten years	in 1 Case
Eleven years	in 1 Case
Twelve years	in 1 Case
Not stated	in 1 Case
Total	40

It will be noted in this table that the paralysis appeared in thirty cases after the disease had existed from a few months to five years. Thirteen occurred between one and two years, while one each occurred ten, eleven and twelve years after the disease was first discovered.

TABLE No. IV.

First Symptom of Paralysis.

More or less sudden loss of power	in 15 Cases
Gradually increasing weakness of lower ankle extremities	in 13 Cases
Exaggerated patellar tendon reflex with ankle clonus	in 9 Cases
"Numbness"	in 1 Case
Not stated	in 2 Cases
Total	40

It is not always easy to determine, among the class described in this paper, just when the first symptom of paralysis occurred. A slight weakness or even an irregularity in gait is not always noticed unless it is preceded by some distinct traumatism. In one case occurring in private practice I am sure the disease (seventh cervical and first dorsal) had existed for at least two years without detection—a somewhat sudden attack of partial paralysis causing the attending physician to examine the spine. This is especially true of the seventh cervical and first dorsal, but much less so of the mid-dorsal and dorsal lumbar regions.

TABLE No. V.

Relation of Abscess to the Paraplegic Symptoms.

Cases with abscess	12
Cases without abscess	28
Total	40
Apparently benefitted by opening of abscess	4
Apparently not benefitted by opening of abscess	1
Apparently having no effect	7
Total	12
Of the seven reported as having no effect:	
Disappeared spontaneously	4
Appeared before the paralysis	2
Appeared after the paralysis, was cured	1
Total	7

This table seems to need no special comment, except that the "twenty-eight cases without abscess" might better be written "twenty-eight cases without external evidence of abscess." No doubt in some of the twenty-eight cases a small abscess existed, which either became absorbed or encysted.

TABLE No. VI.

Amount of Deformity.

Slight	14
Marked	21
Great	5
Total	40

Dr. Myers calls attention to the fact "that the character of the kyphosis does not affect the paraplegia." The table would seem to indicate the same conclusion. The paralysis was no greater in the cases with "great" deformity than in the "slight" cases. It was sometimes quite the reverse.

TABLE No. VII.

Showing the Variety of Paralysis, with or without Bladder or Rectal Symptoms.

Simple motor paralysis	24
Motor and sensory paralysis	16
Total	40

It is unusual for a sensory paralysis to develop first. There is usually a slight motor invasion, followed in some cases by a sensory disturbance later. The primary invasion of sensory symptoms, especially if accompanied by much pain, always suggests malignant disease—a benign tumor, or some traumatic invasion of the spinal canal.

TABLE No. VIII.

Duration of Motor Paralysis after Treatment was begun. There were in all 24 cases of motor paralysis.

Of the CURED cases the paralysis existed

Three months	in 1 Case
Six months	in 4 Cases
Eight months	in 4 Cases
Nine months	in 1 Case
Ten months	in 2 Cases
One year	in 2 Cases
Sixteen months	in 1 Case
Two years	in 4 Cases
Two years and two months	in 1 Case
Two years and three months	in 1 Case
Total	21

Of the Cases STILL UNDER OBSERVATION, the paralysis has been present,

Two years	in 1 Case
Six years	in 1 Case
Ten years	in 1 Case
Total	3
Total	24

This table, which shows the length of time the patients received treatment for the paralysis, is a very important one. It will be considered in the body of the paper.

TABLE No. IX.

Duration of Sensory Paralysis (after treatment was begun) either with or without Bladder and Rectal Symptoms.

In the cured cases the paralysis existed

Two months	in 1 Case
Three months	in 2 Cases
Five months	in 1 Case
Eight months	in 1 Case
Twelve[1] months	in 1 Case
Eighteen months	in 3 Cases
Two years	in 1 Case
Two[1] years and three months	in 1 Case
Total	11

STILL UNDER OBSERVATION.

One[2] and one-half years	in 1 Case
Three years	in 1 Case
Three[2] and one-half years	in 1 Case
Six years	in 1 Case
Died	1 Case
Total	5
Total	16

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[1] These two had sensory paralysis without bladder or rectal symptoms.

[2] These two have recovered from bladder and rectal symptoms, but have a remaining persistent motor paralysis.

Like Table No. VIII., this will receive attention later in a discussion of the results of treatment.

TABLE No. X.

Giving the length of time in which Paralysis developed after Treatment of the Pott's Disease was commenced.

The paralysis occurred, after treatment was commenced, in

One month	in 1 Case
Two months	in 5 Cases
Four months	in 1 Case
Six months	in 3 Cases
Ten months	in 1 Case
One year	in 3 Cases
One year and six months	in 1 Case
Two years	in 2 Cases
Three years	in 1 Case
Three years and six months	in 3 Cases
Four years	in 2 Cases
Five years	in 1 Case
Ten years	in 1 Case
Not stated	in 2 Cases
Total	27
Paralyzed when admitted to Dispensary treatment	13
Total	40

One might infer, if mechanical treatment could cure paraplegia, that it might more easily prevent its occurrence. But this is not wholly true. In fourteen cases the patients had been under treatment one year or less when the paralysis ensued. But these patients lead a rough life—go to public school and play on the streets and are subjected to traumatism at almost every step. In one case, paraplegia developed ten years after treatment was commenced, though it must not be thought that *active* treatment and supervision was kept up all this time. Even in the best and most carefully treated cases, paraplegia may occur. Many factors bear upon this point—our uncertain knowledge of just what bony changes are going on, and whether there is a closely retained abscess near the canal, are among them.

TABLE No. XI.

DEATHS.

Of general tuberculosis (while still paralyzed)	1
Of general tuberculosis (after complete recovery)	1
Of pneumonia (after complete recovery)	1
Total	3

Only one death occurred while the patient was still under treatment for the paraplegia. Both the others had recovered and had been free from paraplegia for some months.

TABLE No. XII.

RESULTS.

Cases treated of simple motor paralysis	24	
Recovered	21	
Still under observation	3	
Total		24
Cases treated of sensory and motor paralysis	16	
Recovered	11	
Died or still under observation	5	
Total		16
Total		40

SUMMARY.

Treated, 40; recovered, 32; under treatment, 8.[1]

Several conclusions may be drawn from a study of these cases and their histories as shown in the tables.

First of all comes the fact, proven by these results, that Pott's paraplegia, while a condition which demands prolonged care and attention, can ordinarily be cured in a large percentage of cases by careful mechanical treatment. And this being admitted, the question as to the detail of the mechanical support is important.

Whatever the treatment may be, it is of the utmost importance that the spine should be thoroughly "splinted." Simple recumbency with or without traction by weight and pulley from the head and feet, is not sufficient. One must bear in mind, especially in dorsal disease, that even respiration is a traumatism, and that any unnecessary movement is contra-indicated. The head traction is useless in my experience, except perhaps in high up cervical disease. The plaster-of-Paris jacket, while affording some support, fails to make the uniform pressure along either side of the spine. As indicated on a previous page, the simple antero-posterior support, made of malleable steel, and which can be adjusted with absolute accuracy, forms the most rational method of protection. This can be so changed in its bearings and so modified as to its pressure that adequate support can be secured which is neither uncomfortable nor irksome. A water bed adds greatly to the efficiency of the treatment, but an ordinary mattress well supported by boards underneath is generally all that is needed.

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[1] Of the 32 recoveries, 15 walk well with exaggerated patellar tendon reflexes, 5 walk well with normal knee jerk, and of the remaining 12 we have no record of the reflexes.

We come now to a general consideration of the facts brought out by the tables.

Of the forty reported cases thirty-two recovered, three died, one while still paralyzed and under treatment, and one after recovery, both from general tuberculosis. One died from pneumonia after a full recovery from the paraplegia.

Of those remaining under observation, several, perhaps one-half, have a reasonable hope of recovery.

I might say here that in all the cases reported only one had bed sores and none had cystitis. Indeed, in children no attempt was made at catheterization and no serious inconvenience resulted. Perhaps this is the reason why there was such a uniform exemption from cystitis.

And I might say further that I have purposely avoided referring to the questions raised by the existence or absence of anæthesia, hyperæsthesia, etc., in these remarks. The histories were too indefinite in this respect—because in some cases only gross tests were made, in others none at all.

The question may be asked, What shall be done with the four patients still under observation, who have been paralyzed from six to ten years? Shall mechanical treatment be continued indefinitely, or are they proper subjects for laminectomy?

It seems to me that in most if not all these four cases, degeneration of the spinal cord has occurred, and that an operation would be useless. It will be noticed that the longest duration of the paralysis, where recovery took place, with both the motor and sensory cases, was two years and three months, and that in all the recoveries only six got well after one year of treatment. Throwing out of the question incidental matters, such as variation in the home care, the resistance some children inevitably make to restraint, etc., it would seem that after one year has elapsed the chances of recovery diminish under mechanical treatment.

And this raises the question as to whether we are able to make an exact diagnosis in the earlier stages of paraplegia? I think we can—that is, we can judge very accurately as to the condition of the cord and its membranes, but I think we are unable to make an equally certain diagnosis as to the seat and extent of the bone lesion, the existence or non-existence of matter under pressure. This is proven in a case which occurred in St. Luke's Hospital in 1876, under the care of Dr. Abbe. In this case an autopsy showed disease of the eighth dorsal vertebra, which had become necrotic, and a very sharp spicula of bone had been thrust forward into the canal, producing an almost complete section of the cord. Up to a certain point the man's symptoms had not been unfavorable, and the sudden onset of urgent symptoms was unexplained until after his death.

It seems to me that we are fully justified in saying that a case of simple motor paralysis should not be subjected to laminectomy. As long as the sensory tract is unaffected, surgical interference is uncalled for. And even if the sensory area is involved with vesical and rectal paralysis, the chances are very favorable for recovery without operation. If my tables are correct, the chances for recovery are at least 11 to 5 in the sensory cases, in dispensary practice, and the proportion of recoveries are much greater in private practice. In the motor paralysis the chances are 21 to 3, in dispensary practice, and the list of failures ought to be almost nil in private practice.

It is important to be able to recognize the approaching paralysis, and the condition of the reflexes is a valuable aid in this matter. Dr. T. Halsted Myers in his valuable paper[1] says: "The first indication of paraplegia is the exaggerated knee-jerk." In nine of the cases here reported the members of the staff recognized the approaching paralysis by the existence of an exaggerated knee jerk, associated with a slight ankle clonus. It is always a safe rule to at once place a patient with Pott's disease, with these symptoms, in bed with or without a suitable support. But a simple exaggerated knee jerk—or even quite a marked condition of this kind—does not necessarily indicate an approaching paraplegia. In chronic spondylitis of any of the vertebræ above the first lumbar, the patellar reflex is often greatly exaggerated, and it may remain so almost indefinitely, with no subsequent symptoms elsewhere. But add to this even a slight clonus at the ankle and the case at once assumes a different aspect.

As a matter of illustration, I may mention the condition of a patient now under observation, a child of 5 years, with disease at the second dorsal. A month ago I noticed an exaggerated patellar reflex following a simple, but somewhat prolonged cough from bronchitis. The knee jerk, already slightly exaggerated, became much more so, and a slight ankle clonus appeared. The patient was placed in bed, and strict recumbency with sand bags at either side of the body, was maintained. For two weeks no improvement occurred. I then applied a light spinal brace without a chin piece, adjusting it carefully to the outline of the spine. In one week the clonus became modified and soon wholly disappeared. If this were an exceptional case, it might not be worth recording. But I have had the same experience on several other occasions, and I venture to record it as a not unimportant clinical fact.

The point I wish to emphasize is that the prodomata of the paraplegia of Pott's disease are generally well marked, and ought to be easily recognized.

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[1] Op. cit.

Another clinical fact might be recorded here: Whooping cough, especially if it occurs in connection with dorsal disease, is very apt to be followed by not only a rapidly increasing deformity, but it may also be followed by a persistent paraplegia. Any prolonged cough, persistent hiccough, or severe sneezing endangers the cord, especially in dorsal disease.

The relation of abscess to the paraplegic symptoms is very interesting. In a case early in my experience, involving the 7th to 10th dorsal, a somewhat sudden paraplegia was followed by the appearance of a large inter-costal abscess. Aspiration produced almost immediate relief, and a cure soon resulted. I have been interested in similar cases ever since, but the relation of abscess to the paraplegia is not a certain one. Table No. V. will illustrate this very clearly. Of the 40 cases, 12 had abscesses, and 28 had no external evidence of abscess. Four of the twelve were apparently benefitted by opening the abscess, and one was not benefitted; and in seven instances the abscess had no apparent effect on the paraplegic condition. In two cases the abscess appeared before the paraplegia, and in one case abscess appeared after recovery.

The location of the disease is very suggestive. Of the 40, 37 had a single focus of disease; 2 in the cervical region, 3 in the cervico-dorsal, 11 in the upper dorsal, 14 in the mid-dorsal, and 3 in the dorso-lumbar. Paralysis from the lumbar disease is very rare in my own experience. Dr. Myers[1] found 18 in 218 cases.

The remaining three had double foci of disease. One, upper dorsal and lumbar; one, cervico-dorsal and dorso-lumbar; and one, upper dorsal and dorso-lumbar. In each

[1] Op. cit.

one of the three cases it is probable that the upper focus of disease produced the paralysis.

It would seem from Table No. III., that paraplegia may develop at any time in the history of the disease, and I have known patients who are supposed to be well to have paraplegia. In one of the cases reported upon to-night, paralysis occurred before any deformity existed. My table shows that it may occur as early as a few months after the disease has been recognized, or as late as 13 years after its first appearance. It is safe to say that no patient is free from paraplegia until the tubercular process has entirely stopped and the vertebræ are consolidated, though of course, as the time passes, the danger of paralysis becomes less.

As regards the duration of the motor paralysis, under mechanical treatment, in those who recovered, it will be seen in Table No. VIII. that it existed in periods varying from 3 months to 2¼ years in 21 cases. The average duration of the simple motor paralysis was about 7 months.

The duration of the sensory paralysis, with bladder and rectal symptoms in 9 cases, varied from 2 months to 2 years. The average duration of the paralysis was 10 months.

In two cases, with sensory paralysis, without vesical or rectal symptoms, the duration of the paralysis was 19½ months.

Of those who still remain under observation—8 cases in all—two have recovered from severe and prolonged bladder and rectal symptoms, but have remaining a persistent motor paralysis.

Of these eight cases, four represent a series of cases to which full reference has been made, the question being: Are they fit subjects for laminectomy?

In closing my remarks, I would like an opportunity to record before the Society a fact which I think has not been referred to before.

Pott's disease, in the dorso-lumbar region, as well as in confirmed rotary lateral curvature in the same region, may seriously interfere with, or may wholly arrest, menstruation. I have the records of four cases which bear upon this point. The first one is a woman now about 48 years old. I first saw her over 20 years ago. Up to 20 years of age she had been apparently a healthy woman, menstruating regularly. At that age she developed Pott's disease involving the last three dorsal and the first and second lumbar vertebræ. The disease progressed rapidly, with the formation of an intra-pelvic abscess on the left side, which gave rise to no trouble and was gradually absorbed. At 25 she had ceased to menstruate, although she had maintained good general health throughout the entire time, and was to all external appearances a well-developed woman. She has not menstruated since.

The second case, a woman of 25, when I saw her first about 15 years ago, had Pott's disease involving the 9th and 10th dorsal and the first lumbar vertebræ. The disease developed before adolescence. She never menstruated, although the mammary development was not below the average, and she looked like a well-developed and well-nourished woman.

The third case, involving the 12th dorsal and the 1st and 2d lumbar vertebræ, aged 35, in all external respects a fully developed woman, menstruated at intervals and very irregularly for a few years (the disease developed at 15), and after 25 or thereabouts, she menstruated only once in two or three years. And this week I examined a woman aged 43, with very marked rotary lateral curvature in the dorso-lumbar region, the curvature having existed since childhood, who has menstruated only 6 or 7 times in her whole life. In other respects, and aside from her deformity, she has been a healthy, active woman.

In several other cases I have found the menstrual function extremely irregular in women who have had Pott's disease in the dorso-lumbar region early in life.

I have never found a similar condition in Pott's disease above the 10th dorsal, though I have carefully questioned many patients whom I have examined.

These facts I have publicly mentioned on several occasions; before the Orthopædic Section of the N. Y. Academy of Medicine over five years ago, and in my lectures at the N. Y. Orthopædic Dispensary and Hospital during the past twelve years. I deem them worthy of a more permanent record and take pleasure in bringing them to your notice.

PART II

Hip and Ankle Joint Diseases

THE ETIOLOGY AND PATHOLOGY OF CHRONIC JOINT DISEASE

A Series of American Lectures, Edited by E. C. Seguin, M.D., Vol. iii.,
No. vi. Putnam, 1878

Accompanying chronic diseases of the articulations, there are several interesting and instructive phenomena to which I would like to call your attention, and I wish especially to refer to them, as pertaining to the etiology and pathology of the lesion. In adopting this course, I propose, so far as possible, to give the results of my own observations, which are based upon an experience gained in following many cases in the service of the Orthopædic Dispensary and Hospital, supplementing them, as occasion demands, by illustrations drawn from other sources. The questions involved are very suggestive, and the limits of this lecture will not permit me to do full justice to the subject. I shall therefore strive to be both concise and practical in my remarks.

In order to avoid ambiguity and repetition, I wish to state at the outset, that in using the phrase "chronic joint disease" in my remarks, I refer, unless otherwise stated, to that form of articular disease, of which we may take morbus coxarius as a type. The various terms "arthritis," "synovitis," etc., all have a more limited meaning than the comprehensive appellation I have chosen to designate the lesion under consideration; for if I interpret the matter rightly, we mean far more in saying that a patient has "joint disease," than by simply stating, for instance, that the individual has an "arthritis." The former carries with it a sense of chronicity which does not pertain to the latter.

From whatever standpoint we may regard the etiology of chronic joint disease, its peculiar clinical history, and its many unfavorable sequelæ will be generally admitted. As usually met with, the symptoms of this form of articular disease are indicative of a progressive lesion, chronic in its nature, not preceded by any acute symptoms, and unaccompanied in its earlier stage by any marked febrile excitement. It is difficult to control, whatever be the treatment employed. It not unfrequently terminates in suppuration,—ultimates in various deformities and atrophy of the limb,—and death ensues in a certain number of cases. The insidious character of the initial symptoms, as followed by the gradual development of the limp, as in hip joint disease, the inconstant character of the pain, the progressive debility of the limb, the impaired mobility of the articulation, the atrophic disturbance of the muscles and their peculiar spasm, are sooner or later succeeded by the more expressive indices of the lesion, which, as it finds expression in deformity and suppuration, gives rise to results which are familiar to all. It is a disease too, in the very large proportion of cases, of early life, occurring during the period of development, when nature's powers of resistance to the invasion of a chronic disease are at their maximum; but also where in certain conditions of the constitution an exaggerated physiological condition is readily converted into a pathological one: and where, a slight lesion being established, a retrograde takes the place of recuperative action. It is not in short an *acute* disease in any sense, but one developed in obscurity, and progressing through all its stages attended by symptoms which can only be called *chronic*.

The definition given by Prof. Gross, to chronic inflammation, so well answers that which might be applied to a chronic joint disease, that I quote it here,—"chronic inflammation, however provoked, is generally tardy and sluggish in its movements, creating little constitutional disturbance, but not on this account the less surely and effectually undermining the part and system. In the acute variety, the action is rapid, bold, daring; suffering is severe, and constitutional response loud and unmistakable. In chronic inflammation, on the other hand, the symptoms are, as already stated, often obscure, if not absolutely masked, and the embers of disease never break out into open flame. The disease may continue weeks and months, now stationary, smothered or apparently receding; now advancing and seeming almost ready to assume the acute type." Describing the effects of chronic inflammation, Dr. Gross further says,—"The most common and important of these changes, are suppuration, ulceration, softening, adhesion, contraction, induration and enlargement."[1] Though this is the definition of chronic inflammation, accompanied by a description of its effects, I doubt if the history of chronic joint disease could be better described in so few words.

Subcutaneous local lesions, having traumatism as a cause, and involving non-vital organs (especially the upper and lower extremities) are, as a class, the simplest, so far as treatment and results are concerned, that fall to the lot of the surgeon. They are simply *local lesions*, which run an acute course, and even if neglected and abused, rarely terminate in suppuration. This accident is liable to occur only in those conditions, where either from hereditary or acquired causes, there exists an impaired nutrition. These purely local lesions are to be regarded as likely to be followed by serious consequences, as a class, under such circumstances as those which Mr. Paget describes in his "Calamities of Surgery,"[1] where a slight lesion may be followed by exaggerated results, and the minor *cause* may easily become the *occasion* for retrograde, rather than a reparative process. Examples of these conditions are met with in daily practice, and the wards of our hospitals afford ample opportunities for demonstrating them. So far as the articulations are concerned, perhaps the typical lesions excited by traumatism, at least the most common, are those produced by sprains. We know how severe they may be, and how chronic they may become. The joint may be stiff and useless; extra, or even intra-capsular adhesions may form as the result of the inflammation excited, by sometimes comparatively slight, and at other times very severe injuries; or the joint may become practically useless, exquisitely sensitive on pressure at certain points, and yet suppuration rarely ensues. None of the characteristic symptoms of chronic joint disease supervene in my own experience, nor has it in the experience of those I have consulted. The pathology of this condition has been recently very concisely described by Prof. R. O. Cowling. I quote the following from his paper,—"Languor in the circulation is the prominent feature and the key to most of the conditions. There is mal-nutrition, local in its origin. Plastic deposits resulting in the inflammatory stage, or slowly gathering as the result of the chronic congestion, are not taken up. Tendons, ligaments, and aponeurosis are shortened by continued disuse. The bodies of the muscles waste and then contractility is lost. The nerve structure has not been repaired. New fibers have been developed in adventitious tissue, and they alike cry for good blood and moving blood. With such a condition of affairs, loss of function, pain, and tenderness naturally continue."[1]

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[1] *A System of Surgery*, by Prof. S. D. Gross. Vol. i, p. 105.

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[1] *Clinical Lectures and Essays*, by Sir J. Paget, page 51.

In chronic joint disease the condition is far different. There is no plastic material thrown out; no adventitious tissue is formed. The mal-nutrition is not local in its origin, but general, and in the place of the higher inflammatory products a "regressive metamorphosis" occurs, that ultimates in the formation of a degenerate pus, filled with cheesy flocculi and necrosed cellular tissue. The sprain is the legitimate result of traumatism. The joint disease frequently does not require any blow or fall to excite the local symptoms of the lesion, the morbid elements of which antedate the injury even if it were received. The whole animal economy calls for "good blood and moving blood," not the joint alone. The entire system needs assistance in establishing a normal reaction in the part which is but indicating the general condition. In other words, the joint lesion may frequently be called a symptom; a symptom of sufficient importance however to require extensive local treatment.

If we take, for example, a case of chronic joint disease at the earliest manifestation of the local symptoms, and treat it *locally*, as we would a fracture or a dislocation, can we assure ourselves that we will arrest the disease? Can we even feel certain that pus will not form? I do not mean to disparage local treatment in joint disease when I say that we cannot. On the other hand, a local lesion in a healthy individual requires no constitutional treatment, and even in the severer injuries of the articulations,—such as fracture near a joint, or a dislocation, where the nutrition of the articulation is materially interfered with locally, and its ligamentous and capsular structures lacerated, the symptoms of a chronic suppurative inflammation rarely follow. To summarize,—Experience proves that traumatism excites acute lesions only, as a rule. In those constitutions strong enough to resist and repair the injury, these acute troubles soon subside; under reverse circumstances, they are apt to be followed by a chronic form of inflammation, which may end in suppuration. As applied to joint disease, traumatism is not a prolific cause of chronic inflammation, except in the conditions noted, and then it acts as an *exciting* cause only. We may state still farther. Traumatic joint lesions (excluding incised wounds of the capsule) are not very frequently seen, unless we accept sprain and dislocation as being lesions of this character. When, however, these typical traumatic joint lesions occur, they present symptoms that are unmistakable. They no more resemble the ordinary forms of chronic joint disease, in their course and history, than a fracture resembles a chronic osteitis. The comparison may be carried still further. Traumatic joint lesions, as a class, recover (as compared with the chronic) rapidly, while chronic joint disease runs a course of years. Acute joint disease (synovitis), does not usually, in my experience, degenerate even in strumous individuals into a chronic form of inflammation, while chronic joint disease has an antecedent history, which, like the prodromata of acute hydrocepalus, is vague and indistinct, and it is not until some expressive local symptom presents, that the disease is really recognized. It is chronic from its very outset, and depends on a predisposing condition which has been variously defined and vaguely stated by different authorities. I cannot describe it better, than by stating that the condition is accompanied by a diathesis, that is characterized by an abnormal vulnerability which depends on an acquired or hereditary predisposition to chronic inflammation, the tendency of which is to a regressive metamorphosis, rather than to the formation of plastic elements.[1]

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[1] The Pathology and Treatment of Sprains." *Louisville Medical News*, May 19, 1877.

In the great majority of children that have presented for examination, both in Dispensary and private practice, with hip joint disease, *e.g.*, the first symptom noted by the parents has not been pain—that which we would expect to find were the cause traumatic—but a limp. This limp would be preceded by a fall in many of the cases (for how many youngsters are there who have escaped a severe fall), to which the parents attached a greater or less importance. But in all cases, with a few exceptions, the injury was followed by no acute symptom, nor any manifestation which could be traced to a definite local lesion. In those cases where I have had the opportunity to examine the patient in this stage of the disease, I have found no constitutional disturbance, the thermometer indicating a normal degree of temperature. Pain generally, but not always, can be excited by some of the various movements of the articulation, which are resisted in some of the normal arcs of motion, by a peculiar spasm of the muscle. The patient presents a peculiar attitude, bearing his weight on the sound side, flexion of the joint occurs, and with it other symptoms which depend on the locality affected. This obscure condition is not unfrequently simulated by some slight trouble, or acute peri-articular lesion. On some future occasion, I shall dwell upon this point in detail. I need only say now, that if in such a condition as I have described, a high temperature (100.5° or over) exists, do not make a diagnosis of chronic joint disease. Suppuration in the early stage of any chronic disease is not likely to occur. If you have a high temperature, the chances are that your "joint disease" will disappear in a few days or weeks, and without the aid of any extension or other mechanical support. All the prominent symptoms may be present, such as flexed and an adducted thigh (if the hip be suspected) muscular rigidity preventing free movement of the joint, flattening of the natis, obliteration of the gluteo-femoral crease, pain on concussion or pressure, etc.; but a high temperature indicates just that condition which does not exist in chronic joint disease, unless active suppuration be present. 99° to 100° (sub lingual), I do not, in children, regard necessarily as an abnormal temperature, though the latter is always suspicious. Still, I have watched and followed several cases where it existed for some weeks, and in one case months, without any sequelæ. It is a safe rule never to make, especially with but one examination, a diagnosis of chronic joint disease, in the early stage, where the temperature is 101° or over. A high evening temperature, with a fall to 99° or thereabouts in the morning, may indicate (and in joint disease generally does indicate) suppuration. But accompanying this condition are other symptoms which I cannot here stop to point out.

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[1] Modified from Birsch-Hirschfeld's definition of Scrofulosis, Vol. xvi. of Ziemssen's *Cyclopædia of Medicine*, page 761.

I will relate, as illustrating the diagnostic value of a high temperature under the circumstances just mentioned, the history and result of a case which I have recently seen.

Mary Smith, aged 10, was admitted to St. Luke's Hospital, on October 16, 1877, during the service of Dr. J. L. Little. I happened to be making my usual daily round when Dr. Little examined the patient. He asked my opinion regarding the case, the history of which was as follows: Several months ago the patient had typhoid fever. Eight weeks before her admission, while convalescent, she fell from the bed to the floor, a distance of about two feet, striking upon her right hip. Several days after she complained of pain in the region of the hip, and a few weeks later, the mother noticed a considerable swelling, and great tenderness about the trochanter major. This increased up to the time of admission. Two weeks ago, the patient, who had been limping about, became unable to walk alone. On admission, there was evident a considerable swelling in the region of, and below, the trochanter major, with distinct fluctuation. The limb was flexed on the pelvis at 30° and slightly adducted. Upon examining the limb and joint, I found that the movements of the latter were very limited, flexion of the thigh stopping at 45°. Adduction and abduction were very slight. Rotation limited, extreme attempts at motion in this direction being accompanied by pelvic movements, and extension could not be executed beyond the 30° of flexion. Any sudden motion of limb caused pain. Pain not increased by pressing articular surfaces together. The patient was very greatly emaciated, and the thighs equally atrophied, were about eight inches only in circumference.

A hip joint lesion was so closely simulated by the objective symptoms, that upon a casual inspection, I felt inclined to diagnose a morbus coxarius. A careful examination, and a consideration of the history, caused me to think differently, and the temperature being 102°, I at once decided that it was an acute extra capsular lesion, an opinion also which Dr. Little held prior to my examination. The subsequent history of this case, the progress of which I noted frequently, I append in full, from notes furnished me by Dr. Alonzo Blauvelt, House Surgeon.

Oct. 17. Patient etherized, and abscess opened freely by Dr. Little, under Lister. About a pint of thick healthy pus evacuated. A digital examination failed to connect the abscess with the joint. Drainage tube inserted and Lister's dressings applied.

Oct. 30. Wound has been dressed every second or third day. Lister's antiseptic precautions and dressings being carefully followed. At this date very slight discharge. Drainage tube removed. Temperature has ranged from 99° to 101.5° in A.M., and from 101° to 103° in P.M. Patient able to move limb in every direction with very little pain. Tonic treatment ordered.

Nov. 15. Wound dressed only about once a week since last record. Entirely healed except a small sinus made by drainage tube. Probe can be passed about 3½ inches upwards, and a little forwards. Temperature lower. Bals. Peru injected in sinus, and compress over parts. Good motion of joint without pain.

Nov. 30. Sinus entirely healed. Patient allowed to walk around. Temperature nearly normal. Has increased much in flesh. General health improving rapidly.

Dec. 6. Limb can be moved in any direction without causing any pain whatever. Joint motion normal. Patient walks about ward the entire day, with a scarcely perceptible limp. Temperature normal.

I am indebted to my colleague, Dr. Charles McBurney, under whose care the patient now is, for permission to report this interesting case.

To return to the limp. I stated that it might be accompanied by pain. The pain however like the limp is indefinite and vague, and sometimes remits, and not unfrequently changes its location. The parents are apt on these accounts, to call it a "growing pain." But the limp returns. The pain becomes, perhaps suddenly, persistent. It disturbs the patient at night, and is sometimes accompanied by a cry resembling that which occurs in acute hydrocephalus. The child does not fairly awaken during these paroxysms. But the cry is peculiar, and the incoherent mutterings are not remembered even if the patient be fully awakened. If the disease be located in the hip, the thigh begins to flex; the joint motions are limited; the muscles begin to waste, and the patient now walks very badly. Symptomatic fever sooner or later ensues, with a temperature curve that reaches its maximum in the evening, and that touches nearly normal in the morning. Pus is forming, though we may not have external evidence of it for months, when a "cold abscess" will appear. Finally, the patient suffers excruciating pain; the already weakened constitution sympathizes very evidently, the face assumes a care-worn, aged look, and a progressive general atrophy of its various tissues, indicate the extent to which the system is impaired. The abscess discharges copiously an ichorous, shreddy pus. The probe reaches carious bone through the sinuses thus established, and in some cases the patient succumbs, either from exhaustion, or from amyloid degeneration. The very great majority of cases of chronic joint disease, properly treated, do not terminate fatally, but the disease in any event, whether terminating in suppuration and caries, or whether arrested prior to these unfortunate complications, is a very chronic one, requiring a long and persevering treatment, and the result is not, as a rule, a symmetrical limb with good joint motion, but a shortened, atrophied member, useful, no doubt, and strong, but not perfect.

I have tried not to overdraw this sketch, and as illustrated by the average cases of chronic joint disease which have applied to the Dispensary, it is not exaggerated. I wish to ask if it resembles, in any important respect traumatic lesions as applied to the same structures? Does a disease, so essentially progressive, so persistently chronic, so slowly responsive to both local and constitutional treatment, develop from a simple local cause, (so frequently guessed at by the parents, and adopted by the surgeon on this authority,) and which, passing through its primal stages almost unnoticed, or at least, without constitutional disturbance, progresses slowly though surely, until suppuration sends the mercury up to 101°–105°?

Joint disease may be either acute or chronic; when acute, it usually affects the synovial membrane; when chronic, I believe it most frequently has its origin, especially in the hip joint, in the osseous structure (epiphysis). The former, sometimes difficult to control, and generally idiopathic, passes through its various stages, and in my own experience, as before remarked, even in strumous children seldom develops into the typical chronic joint disease. Acute inflammation of the synovial membrane is not very frequently seen in the hip-joint. The knee is its favorite seat. When, however, it does affect the hip, the lesion can scarcely be mistaken for any other condition. A high temperature, an acutely sensitive joint, a rapid pulse, a "swift atrophy of the muscles," the whole generally preceded by a chill, and with the local symptoms pointing to the affected articulation leave very little room for doubt. A typical case of this somewhat rare affection, and presenting some interesting and peculiar features, occurred in my private practice not long ago. The history is as follows:

On Sept. 10, 1876, I was called to see, in consultation with Dr. A. W. Catlin, of Brooklyn, a little girl, Lucy H., aged 10. Two or three days previously the patient had been to Coney Island, and in returning late in the evening complained of feeling very chilly. She went to bed immediately upon her arrival home, feeling somewhat ill. During the night she was seized with a violent pain in the hip, and fever rapidly supervened. When examined by Dr. Catlin the following morning, the hip joint (right) was found to be very sensitive, both upon pressure and motion. The thigh was flexed, *abducted* and apparently *longer* than its mate. Temperature 102.5°, pulse rapid. Counter irritation and antiphlogistic measures were employed. The malposition remaining, and the lengthening of the limb being not only apparent but *real*, we met in consultation as above stated.

Upon examination, a most peculiar condition of affairs was made manifest. The rational symptoms all pointed to an acute disease, but the physical strongly suggested a dislocation. The pelvis was tilted downward on the affected side, the trochanter was apparently displaced downward and backward, an actual depression existed in the femoral region, and the limb rotated outward, flexed and abducted, was after careful measurement found to be three-eighths of an inch longer than its fellow. Accompanying this condition, was a marked atrophy of the gluteus maximus and quadriceps extensor cruris. Slight motion produced very severe pain. The temperature was still high (102°) and all the indications of fever were present. The acute symptoms were easily accounted for, and pointed to an acute synovitis of the hip-joint. But how with these symptoms were we to account for the actual lengthening? In view of this latter fact, a diagnosis other than acute synovitis was not made.

Two days later we met again, when the pain having modified to a considerable extent, a more thorough examination was permitted. The lengthening was still present, and the position of the thigh was not changed. Palpation and a careful examination of the joint proved that there was no displacement. I then stated my belief that the lengthening was congenital, and could be thus eliminated from our differential diagnosis, and that the trouble was simply an acute synovitis of the hip, with the characteristic muscular atrophy, as mentioned by Sir James Paget.[1] The position was, I believe, due to the muscular contraction,[2] and the "acute atrophy" of the extensors of the leg occasioned the "depression" in the femoral region. As the patient was of decidedly strumous habit, a careful course of treatment was pursued, which ultimated in complete recovery, including a restoration of the atrophied muscles. This lengthening of three-eighths of an inch still exists. An examination of the father of the child, subsequently, developed the fact that his *left* leg was one-half inch longer than the right, a condition that he said he had often suspected, from the complaints of his tailor.

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[1] *Op. cit.*, page 209.

[2] See my paper on "Reflex Muscular Contraction and Atrophy in Joint Disease," etc. *Archives of Clinical Surgery*, June, 1877.

A bad hereditary history caused us to fear, during our treatment of this case, that the acute trouble might degenerate into a chronic form of inflammation. But no such complication ensued. The recovery, as stated above, was perfect. I have never seen an acute, suppurative synovitis of the hip. When it occurs at the knee-joint, the symptoms are pronounced. Our standard works on surgery treat fully of this condition. The treatment of the joint lesion not unfrequently terminates in amputation. As generally seen, however, it results from incised wounds of the capsule. I distinctly remember an instructive case of this kind, which I saw in the year 1866.

The patient had been discharged from a prominent hospital, because he declined to submit to amputation. I found him in a dark room at the Five Points, surrounded by the most unfavorable sanitary influences. Through sheer pity I promised to attend the boy (he was twelve years old), though without the faintest idea of doing more than easing his path to the grave. The knee-joint (the site of the disease) was wholly disintegrated. Pus had burrowed underneath the fascia in all directions. I made in all, five free incisions in different parts of the limb, one in the upper part of the thigh, and adapted a simple posterior splint. Stimulants and nourishment were supplied by a neighboring charitable institution. The boy made a good recovery, and had, the last time I saw him, an anchylosed joint in a good position. And this recalls another point to my mind which is worthy of remark. It has sometimes occurred that advanced cases of joint disease, exhausted by hectic, and wasted by suppuration, have been discharged from a hospital "to die at home," sometimes at the request of friends. I have seen several such cases afterward with sinuses healed, with health apparent in every expression, and with good, strong, anchylosed joints. I submit these facts simply stating that I have frequently thought, while following advanced cases of profusely suppurating joint disease in hospital practice, that the aggregation of similar cases is contra-indicated. My experience leads me to believe, that, after a lengthened sojourn in a hospital ward, these cases progress much better if removed, provided, of course, that efficient surgical attendance is given, even if the sanitary and hygienic conditions of their homes are not all that could be desired.

I wish now, in contrast with the case of acute synovitis of the hip-joint (Lucy H.) to relate the history and progress of a case of chronic osteitis, involving the same articulation.

Alice Dormer applied at the Orthopædic Dispensary, on June 24, 1875. She was then 5 years old, and resided in Brooklyn. Her father died of pneumonia seven months prior to my examination of the child. He had been a healthy man, by occupation a book-keeper. The mother, who came with the patient, was apparently in good health, but had been subject to attacks of acute rheumatism. The patient has four brothers, all in good health, and one sister "in delicate health." One sister died at eighteen months, of acute hydrocephalus. No hereditary disease on either paternal or maternal side. Joint disease and phthisis unknown in the family history. Upon asking the mother if she knew of any cause for the disease, she stated that she did not. No fall or injury, save the one noted below, known. The history developed was as follows: On the 11th of October, 1874, the patient was playing with her brothers as usual. The next day she limped, complained of "cramps" and pain in the limb. She refused to stand on her leg. In a few hours she was walking around again. From this date to Christmas of the same year, she did not limp when she walked. She had no pain during the day, but at night she would sometimes cry out suddenly, and if, on these occasions, the patient was thoroughly awakened, so that the cause of the crying might be ascertained, she would complain of pain in her thigh and knee, especially if the limb was moved. About Christmas she "caught her foot in the bannister," as she came down stairs. The injury was very slight, and the mother attached but little importance to it at the time. The patient cried, however, and complained of her leg and joint. She commenced to limp the next day, and soon after "a pain in the groin" developed. The symptoms now seemed to demand attention, and the mother sought medical advice at a dispensary. A "sprain or growing pain" was diagnosed. Different remedies, domestic and otherwise, were tried until February 25, 1875, when a Brooklyn surgeon recognized the difficulty, and applied Buck's extension. From that time to the day the patient applied at the Orthopædic Dispensary, she had been kept in bed. The condition of the patient when she applied was as follows: delicate looking, quite anæmic; not well nourished. Dentition regular. Has had none of the diseases incident to childhood, except whooping-cough. The thigh was flexed and adducted, and the joint was very painful on slight motion. Marked atrophy of gluteal muscles on affected side. Obliteration of gluteo-femoral crease. The muscles of the thigh were atrophied also, and the reflex muscular spasm marked. Fluctuation not present. Limbs of equal length.

The patient did not return to the Institution for treatment. An instrument was applied by the family physician in Brooklyn. The mother states that the child grew gradually worse. On October 17th, of the present year, the patient again applied at the Orthopædic Dispensary. Her condition at that time is thus recorded: Very pale and somewhat emaciated. The face shows traces of suffering. The thigh flexed at 90° and adducted at about 30°. Reflex atrophy and contraction very marked—the muscular spasm being so great, that anchylosis is simulated. Temperature 100.5°. Affected limb one inch shorter than its fellow. No pus had formed during the two years that had elapsed since the child was first brought to the Dispensary, or at least none could be detected. Abscess prognosed—child to enter the hospital.

This case illustrates an important point. Obscure symptoms existed before the traumatic element appeared. This, though slight, marked the onset of the more generally recognized symptoms, which might, by many, be deemed those of actual invasion. The disease had been slowly but surely progressing for an indefinite time, and the slight injury received was sufficient, in the peculiar condition of the joint, to produce an effect in no way commensurate with the cause. In my own experience, this is the kind of traumatism which "produces" chronic joint disease.

A brief comparison of the cases of Lucy H., and Alice Dormer will be instructive. In the former there existed a hereditary history that was unfavorable. The lesion was idiopathic, dependant on exposure, partook wholly of an acute character, and terminated favorably. The latter, while pale and anæmic, gave no history in a hereditary sense that was questionable. She was not, however, the victim of traumatism, but nevertheless developed a progressive articular osteitis (epiphysitis) which, in two years, had produced shortening of the femur to the extent of one inch, marked muscular atrophy and spasm, great deformity and suppuration, if it has not already taken place, is likely to occur at any time. The best result that can be anticipated in this case, is a shortened, atrophied limb, with an anchylosed joint. It is possible, should prolonged suppuration occur, that the history may end with amyloid degeneration, and death.

Through the kindness of my colleague, Dr. James L. Little, I have recently had the opportunity of observing critically, a very interesting and rare case, occurring in his service at St. Luke's Hospital, the history of which will further illustrate my belief that traumatism is not an important factor in the production of chronic joint disease. I append the history of the case, accompanied by notes made personally at the frequent examinations, to which I subjected the patient.

Edward Gibbs, aged 5, entered St. Luke's Hospital October 4, 1877. Hereditary history: Maternal grandfather died of phthisis. Paternal side, good, so far as known. Father died from an injury. Mother of patient a thin, anæmic woman, who has been gradually failing in health since the birth of patient. Has lost flesh during the last two years, during which time she has had "a bad cough." The boy in appearance is not strong; has flabby skin, weak eyes; is pale and poorly nourished. Has had measles and whooping-cough, and has recently passed through a prolonged attack of intermittent fever developed in Indianapolis. Has been free from the fever about six months.

On the 23d of September last, the patient fell backward from a wagon in which he was playing, and sustained a severe injury to the hip, which was accompanied by deformity and pain. After a day or two of domestic treatment (the child was boarding at the house of a stranger), and several consultations with physicians, none of whom recognized the difficulty, the child on October 4th, as above stated, was taken to St. Luke's Hospital. Dr. Little found the head of the femur dislocated upon the dorsum of the ilium. The dislocation was reduced by manipulation under ether. On the day following I saw the patient and examined him. There was considerable tenderness about the joint, the movements of which were limited by the pain inflicted. The patient walked with a very evident limp, but complained of no pain in doing so. No rigidity or stiffness marked his gait, and no hesitation was manifested in placing the foot to the floor. His attitude was not unlike that which might arise from a bruise to any part of the limb. Further examination postponed.

Oct. 15. The boy has been moving about the ward at his own pleasure for the past few days, and has daily improved in walking. Examined to-day, especially to ascertain the character of the muscular resistance, and the condition of the joint as regards pain. Patient placed in supine position, and pelvis grasped and steadied with left hand. *Flexion* of thigh slightly resisted; motion nearly normal. With the knee flexed, *rotation inward* gave pain. Motion limited, pain acute, finding expression orally. No reflex muscular spasm, such as is found in chronic joint disease, present. Contraction not persistent. *Abduction* and *adduction* slightly impeded. In prone position, pelvis steadied with left hand, *Extension* resisted quite markedly; not a quick, apprehensive spasm, but a semi-voluntary, elastic, muscular resistance, followed by an exclamation, "Oh!" Flexing the leg at an angle of 90°, with the thigh in the above position, and using the leg thus flexed as a lever to produce *rotation inward*, the same result was obtained. Motion limited, the foot passing through the arc of a circle of about 25°. *Rotation outward*, markedly limited. Patient sleeps well. Has no pain as he plays about the ward.

Oct. 20. Symptoms have all improved. The same tests as above show improved motion, less pain. Patient *runs* about ward without perceptible limp.

Oct. 21. Case seen by Dr. Buckminster Brown, who visited the hospital with me, and attention was called to the history and condition of patient. The slight remaining resistance to joint motion was developed, and noted by Dr. Brown.

Oct. 27. Gluteo-femoral crease, which had been slightly lower on affected side, normal. No flattening of natis. Severe concussion to sole of foot produces no pain or flinching. No atrophy; faradic reaction of the thigh and gluteal muscles normal.

Nov. 24. Very slight, hardly perceptible muscular resistance. Patient runs, jumps, and plays without the slightest limp or complaint.

Dec. 11. Joint motion normal. Patient discharged, cured.

The lessons taught us by this case are both interesting and important. 1. Without a history of dislocation, the condition made evident by examination would have been well calculated to mislead many in diagnosis. In other words, a condition approximating that induced by the injury received, though excited by a less marked traumatic cause, might have been called "incipient hip-disease," and treated as such by, perhaps, extension apparatus, and in a few weeks the patient would have been "cured," when, as the result abundantly proves no such treatment was required. 2. None of the symptoms especially characteristic of the first stage of a chronic joint lesion were present. 3. At no time subsequent to the reduction of the dislocation, was there any disinclination to walk, or hesitation in using the limb. 4. The peculiar and expressive reflex *spasm* of the muscles was absent, though, as in the "hysterical joint," there was a semi-voluntary muscular resistance, which simulated it. 5. The "swift atrophy of the muscles" mentioned by Paget, as following acute synovitis, was not present, nor was the faradic contractility of the muscles reduced. 6. An extensive (and a neglected) traumatic lesion of the hip-joint, in a poorly nourished child, with a very questionable hereditary history, steadily improved from day to day, without any especial treatment, and recovery was rapid and complete. To conclude, we may say that the head of the femur had been dislocated for ten days, and that the capsule of the joint was undoubtedly lacerated and the ligamentum teres ruptured. The patient was only 5 years old, and as likely to develop chronic joint disease as any child that might receive some slight, and almost unnoticed, injury, which months afterward is followed by the chronic joint limp that so frequently precedes suppuration, etc. It was, however, a simple, acute, local lesion and repair took place rapidly. But all the conditions that are ordinarily deemed necessary to excite "joint disease" were present, if traumatism alone could develop it in this particular case. The child was even given the opportunity to irritate the joint by all the exercise he wished to take, which was very considerable. On the other hand, a simple fall, unattended by any especial feature and antedating, in many instances, the initial expression of the local symptoms several months, is frequently made responsible for so grave and so serious a lesion, as chronic joint disease. Unfortunately, especially for our patients, traumatism alone is not responsible for chronic joint disease. If it were, rapid and complete recovery would be the rule,—abscess and progressive deformity the exception.

In treating more directly of the pathology of joint disease, I wish rather to view it from a practical and clinical standpoint, as I have its etiology, and as furnishing indications for treatment, than with the object of contributing an essay upon its minute morbid anatomy. Indeed I have nothing to add to the researches of the able investigators, whose contributions to the pathology of joint disease leave but little to be said. Brodie, Barwell Paget, Billroth, Volkmann, and many others, may be consulted by those who wish especially to study this subject. It is enough for us to know that, for obvious reasons, but few opportunities have been afforded pathologists to record the *initial* changes which occur in the chronic forms of articular disease. The cases on record demonstrate, however, that the primary lesion may occur in almost any of the articular structures, and the facts thus adduced do not, as yet, afford sufficient evidence upon which to base reliable data. Hence it is that we find such a variety of opinions expressed by different surgical authorities regarding these changes. From a clinical standpoint, however, it matters but little where the initial lesion takes place; for in those conditions where chronic joint disease is easily excited, the morbid process sooner or later attacks the neighboring structures, and if it proceed unchecked, the entire joint becomes involved.

An opportunity has been afforded me, through the kindness of Dr. Robert Abbe, to examine the pathological condition of a hip joint, one of the structures of which had become the seat of inflammation in a very peculiar manner.

J. Bruce, aged 30, was, on March 2, 1875, admitted to St. Luke's Hospital, during the service of Dr. James L. Little. The patient was suffering from an aneurism of the femoral artery, for the relief of which Dr. Little ligated the external iliac, on March 9, 1875. On the 22d day the patient died from exhaustion. While performing the autopsy, and in removing the aneurismal tumor, Dr. Abbe accidentally incised the capsule of the hip joint. A few drops of pus followed the incision. After excising the aneurismal sac, a further exploration of the joint revealed the fact that the ligamentum teres had been the seat of acute inflammation. Its surface, highly injected, was of a brilliant scarlet color. About two drachms of laudable pus was found in the capsule. There was no evidence of disease of the cartilage or bone, and the other joint structures were sound. The patient had complained before the operation of pain in the knee, and the thigh was flexed and adducted to a considerable extent. The presence of the aneurism and the pressure it occasioned upon the subjacent parts, were sufficient to account for both the pain and position of the thigh, and no one had suspected the existence of any hip joint lesion. It would not have been discovered, but for the accident referred to, which disclosed the product of inflammation. Regarding the cause of the lesion thus found, it would indeed be difficult to offer a perfectly satisfactory explanation. The tumor did not in any way press upon the joint: if it had we should have looked for a lesion in a different structure. There was no history of direct violence. The limb had been held prior to the existence of the pain in the knee, etc., for many weeks in a *flexed* and *adducted* position. This placed the ligamentum teres upon the stretch, and in this way an irritation was produced that might easily be sufficient to produce the inflammation. This is my explanation of it, and it involves a traumatic cause. The evidences of acute inflammation, and laudable pus—each one (where suppuration ensues) the ordinary result of traumatism—were found at the autopsy.

I do not mention this case as forming the basis of my opinion regarding the early changes which occur in joint disease. On the contrary, my clinical observations, records and studies lead me to different conclusions. I offer this interesting case as a contribution to the, as yet, very incomplete literature of the subject.

While no opportunity, other than the case just mentioned, has been afforded me to examine, the post mortem condition of a diseased joint in the *earliest* stage of the lesion, I have, by watching the development of the symptoms, and noting their sequelæ, in well marked and unmistakable cases, and in obtaining reliable histories from intelligent persons, reached conclusions that are based upon, strictly speaking, clinical experience. It is from this standpoint that I submit my views, selecting such cases as may illustrate the points involved, and stating in general terms the results of my observations, which include the records of several hundred cases that have been under my personal care, both in dispensary and private practice. My personal experience with statistics forbids my presenting them. In joint disease especially, they are of value principally to the compiler, so important are the many modifications of the facts elicited and which cannot be expressed in a few words. Statistics are valuable in reporting results and in classifying absolutely certain facts, such as sex, age, etc. But beyond this, with no universally accepted definition of the many headings necessary to complete a history, statistical tables on joint disease are, at least, unsatisfactory. I have the records of many cases. Instead of presenting tables, I prefer to state my conclusions.

Of the various structures entering into the formation of those joints which are most frequently the seat of disease, the bone and synovial membrane are the most liable to inflammation. The ligaments, from their intimate association with the synovial membrane, are so closely identified with the latter, that it is practically impossible to diagnose an idiopathic lesion of the former. The cartilage, being non-vascular, is rarely, if ever the seat of an idiopathic inflammation, but when, through traumatic cause it becomes injured, serious trouble is apt to ensue. We have then, practically, these two important structures—the bone and synovial membrane, to which we must look for our primary lesion; or, perhaps we will state the case more clearly if we say that in whatever part of the joint the primary change may take place, it is to one of these essential joint factors to which we must look for our symptoms. The early symptoms of either chronic osteitis or synovitis are, generally, clearly defined, and though, as the morbid process advances the distinctive symptoms may become obscure and conflicting, there are, as I shall hope to demonstrate, certain specific indications which are of value as a means of making a differential diagnosis, and upon which, of course, will depend both our prognosis and treatment.

In chronic synovitis, ''we first find swelling and redness of the synovial membrane; it has already undergone some change in the lateral portions of the joint, in the folds and neighboring sacs; its tufts are puffed up, very little elongated, but very soft and succulent; the whole membrane is more readily distinguished from the firm tissues of the capsule, and may be detached with greater facility than normally. At this time the synovia is rarely increased, but is cloudy or even resembles muco-pus. These changes in the synovial membrane gradually increase; it becomes thicker, more œdematous, softer, redder; the tufts grow to thick pads, and in places resemble spongy granulations. The surface of the cartilage loses its blue luster, though it is not yet visibly diseased; but the synovial outgrowths begin to grow over the cartilages from the sides, and to push in between the two adjacent surfaces of cartilage; meantime the capsule of the joint is also thickened, and has acquired an evenly fatty appearance, and is very œdematous; this swelling and œdema gradually extend to the subcutaneous tissue, and to the skin. From this point, the changes in the cartilage claim most of our attention. The synovial proliferations, in the shape of red granular masses advance gradually over the entire surface of the cartilage, and cover it completely, lying over it like a veil; if we attempt to remove this veil, we find that in some places it is attached quite firmly by processes entering the cartilage, just as the roots of an ivy vine cling to and insert themselves into the wall against which it grows; these roots not only elongate, they spread out, and gradually eat up the cartilage, which, when the covering of fungous proliferations is removed, appear first rough here and there, then perforated, and finally disappear altogether; then the fungous proliferation extends into the bone, and commences to consume this; the result is fungous caries." (Billroth's *Surg. Pathology*, page 473.)

I have quoted thus at length from an eminent authority upon the progressive changes which occur in chronic synovitis, for the purpose of calling attention to some of the important symptoms following and accompanying this condition, as it passes through its various stages. This description does not include hydrops articuli, which, as the same authority states "is no more apt to become purulent synovitis than is chronic articular rheumatism."

In the early history of this pathological condition, there may be some pain of an indefinite and vague character. Sometimes there is no pain at all. It does not at least exist as an important symptom, and it is most likely to be developed after prolonged exercise. In my own experience it has never been urgent, sleep being undisturbed by "starting pains," and the incoherent, nocturnal cry. A slight swelling may be noted, principally exhibiting itself, for instance, in an obliteration of the fossa on either side of the patella, if the knee be the location of the disease. The patient is apt to limp slightly, especially after exercise, and the limp thus developed is usually the first symptom noted. No pain being present to locate the trouble in the mind of the parents, and the patient sleeping well, the limp is usually supposed to be a "bad habit." The limp increases, the swelling becomes more apparent, the joint begins to assume a semi-flexed position; pressure over the articulation develops sensitive points. The patient now locates a definite pain, and it is usually at this stage, especially in dispensary practice, that professional advice is sought. The changes in the synovial membrane and the modified secretion resulting therefrom, do not produce an effect which limits the motion of the joint, except in the extremes of movement. The resistance felt in gently urging the distal member of the joint into a position of extreme flexion or extension is not unlike that which would result from a simple distention of the capsule, or the altered and thickened state of the synovial membrane. There is no sudden, apprehensive arrest of motion, caused by a quick, spasmodic contraction of the flexors on the one hand, and the extensors on the other; in other words there is no reflex muscular spasm which arrests movement at a given point, with a quick and decisive check; or, if this muscular spasm exists at all, it is only to a very limited extent. As the disease advances however and the fungous proliferations fairly invade the bone, this muscular spasm occurs to a marked degree, and with it other symptoms appear which will be described later on.

In primary, chronic articular osteitis, on the other hand, which is only too likely to end in caries, the pathological conditions are of a far different character. The lesion involves, to a greater or lesser extent, the epiphysis of one or very rarely two, of the important bones which form the articulation. It is however, like the condition described above, viz.,—*chronic*, and it may have existed, in a sort of latent state for many months, wholly unsuspected, and at a point not immediately contiguous to the articular cartilage. It would certainly seem that this might be true, for the symptoms, once fairly developed, progress as a rule slowly, but with great persistency, and they are very difficult to control. Remission in the symptoms also, is more apt to occur in chronic osteitis than in chronic synovitis. I saw to-day a case, John Reading, æt. 3½, where vague and uncertain symptoms existed all last Summer. He was a little lame at that time, was very restless at night, complained of pain in his hip and knee, and was decidedly disinclined to take exercise. These symptoms all disappeared about September 1st. They again appeared in a couple of weeks, this time accompanied by an inguinal adenitis. Again the child recovered, and ran about without any limp. To-day (November 22nd), this patient, when examined, though limping only very slightly, was found to have a decided reflex spasm of all the important muscles acting on the hip-joint, very limited joint motion, shortening of the limb (a full quarter of an inch), marked atrophy of the muscles of the thigh, which was nearly an inch smaller in circumference than the sound side, and a reduced faradic contractility of the quadriceps extensor and gluteus maximus. The circumference at the calf was the same on either side. But actual pathological observations are wanting on this point, and it is only by calling physiology to our aid that we can reach satisfactory conclusions. In the epiphysis of the long bones, the nutritive processes occur upon which depends the growth of the diaphysis. These epiphyses are very vascular as compared with the dense structure of the shaft, and they are, of all the joint structures, the most abundantly supplied with nervous tissue. Kölliker remarks[1]: "On the articular ends of many bones, such as those of the elbow, knee and ankle joints, I have noticed the nerves to be more abundant than elsewhere." Again (page 338), "In the knee I have seen nerves, even in the true synovial membrane, although in general they are rare, and are most distinct in the large vascular processes." In children these epiphyses are in a condition that approximates hyperæmia; and in that peculiar state which predisposes to chronic joint disease it would seem that their true physiological function was sluggishly performed. I cannot do better in this connection than to quote from Mr. Barwell's excellent treatise on "Diseases of the Joints": "The truth is, that the nutritive activity brought about by the ossifying action in the epiphysis, is very apt to produce a congested condition of the part; and the large bone ends which are found in strumous children are the accompaniment of such tendency to congestion. As the child gets older and stronger, this hyperæmia disappears, and in the great majority of instances is followed by no evil results whatever. It is, however, certain that in a given number of cases the congestion predisposes to inflammation, and the merely passive is followed by an active condition. Thus inflammation may be set up in an epiphyseal end which was previously in an abnormal state, and such in the greater number of cases is the mode in which the disease now under consideration (strumous articular osteitis) commences. All such attacks are in the beginning very slow, and hang for months, perhaps even years, between a state merely of sluggish functional performance and of active disease." My clinical experience fully sustains this last sentence, and indeed, the entire quotation. When this "sluggish functional performance," the initial stage of chronic articular osteitis, terminates in "active disease," the first symptom noticed by the parents will not be pain but, as in chronic synovitis, a limp. In this one respect the two conditions present the same symptom, though the limp of chronic synovitis resembles that of simple joint fatigue, while that which occurs in articular osteitis is more strictly a debility of the entire limb. Indeed, all the symptoms in chronic articular osteitis, from the very onset, show the marked reflex impression which the disease makes upon the nervous system, while those of chronic synovitis are more strictly *local*. These facts, which, so far as I know, have not been noted before, are not difficult to explain when the neural distribution to the two structures, and the conditions described by Charcot, in his work on "Diseases of the Nervous System" as being necessary to produce trophic changes and muscular spasm, are considered.

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[1] *Manual of Human Histology,* Syd. Ed. Vol. i, p. 335.

Sometimes quite early in the history of articular osteitis there occurs this peculiar, incoherent cry at night to which I have referred already as resembling, in some respects, that which occurs in acute hydrocephalus. I have heard it frequently in chronic joint disease when it was very agonizing. The fact, however, that it rarely awakens the patient, and that if he be awakened he knows or remembers nothing of it, leads me to believe that the irritation of the peripheral epiphyseal nerves is both peculiar and profound —for this reflex expression of the lesion occurs during the suspension of the will. Then there is the characteristic reflex spasm of the muscles, a spasm which resembles in many important respects that which follows the remote symptoms of injuries of nerves, as described by Dr. S. Weir Mitchell in his valuable treatise.[1] Accompanying this reflex spasm is a muscular atrophy not dependent upon *disuse*, which is pronounced and which can be demonstrated by the faradic current. And many of the skin troubles which follow during the local treatment of this lesion, and which have been referred, and which are partly due, to the irritating properties of the adhesive plaster, etc., I have sometimes thought were the analogues of the glossy skin and eczema found after neural lesions. I have not time here to amplify this interesting study. I have already stated my views in brief upon this subject.[2] I wish, however, to take this occasion to say that I am indebted to my friend, Dr. E. C. Seguin, for his kind assistance and advice in my investigations.

To return to the symptoms of articular osteitis: The debility of the limb increases gradually, and diurnal pain is, as a rule, first developed by any sudden or unexpected twisting of the limb. Even a very slight movement, for which the patient is unprepared, will cause him to cry out. The "starting pains" accompanied by the osteitic cry become in many instances urgent. The muscles acting upon the articulation, when the joint is examined, show a decided tendency to resist, and a marked spasm (it is more than a "contraction") of the muscles arrests the motion at a given point. In the first stage the resistance may be only felt in the extremes of motion; unlike the sensation imparted when the resistance lies in a thickened or distended capsule, it has a peculiar, apprehensive quality. It is sharply defined, and no effort on the part of the surgeon or patient can overcome it without the use of an anæsthetic. These conditions are best noted in the knee-joint, where an effusion, or a thickened capsule is readily observed, and the existence of muscular spasm easily detected. In illustrating these points I will select two cases of knee-joint disease, both of which are under my observation now in private practice:

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[1] "Injuries of Nerves," by Dr. S. Weir Mitchell, pp. 147–153.

[2] *Op. cit.*

Addie D——, aged five and one-half years; hereditary history shows phthisis on maternal side; child strumous in appearance, waxy skin, etc. Has had very little illness, except that incident to childhood. Was seen in consultation with Dr. S. W. Dana on July 9, 1877. At that time the following history was obtained: Last December (1876) the patient complained slightly of her knees, but the trouble was not severe enough to interfere with the patient's health or movements. No swelling was noticed by the parents, nor was the case considered urgent enough to demand the attention of the family physician. A few days before Dr. Dana brought the child to my notice, he had been called in by the father on account of a swelling which had been observed in *each knee-joint*. An examination proved the existence of a very considerable effusion in either joint, accompanied by an evident local rise of temperature, but with no constitutional disturbance. No pain was produced by movement of the joints, by pressure, or concussion. The child could run and play without inconvenience, limping, or pain. It is not unlikely that limping would have been observed had only *one* joint been affected. Passive movements of the affected joints developed no reflex muscular spasm whatever; an *elastic resistance* limited joint motion in the extremes of flexion and extension. No pain or discomfort at night; no "starting pains"; patient sleeps soundly; no perceptible muscular atrophy. *Diagnosis.*—Chronic synovitis affecting each joint. The bone was excluded from any participation in the disease. As to whether the cartilages were affected is uncertain; probably they were slighty on their articular aspects. Looking forward for an indefinite time, and supposing that the disease proceeds unchecked, both cartilage and bone will become involved, and when the latter has been fairly invaded by the disease, there will occur the sharp nocturnal cry, the reflex muscular spasm, and the other symptoms which I have described as pertaining to articular osteitis.

Willie L——, aged four years, applied at my office on September 11, 1877, with a letter of introduction from Dr. E. D. Hudson, Jr. The patient had been suffering from a chronic disease of the knee-joint for two years. The leg was flexed at an angle of 90° with the thigh, and the head of the tibia had become partially subluxated backward. An examination of the joint revealed a smooth and glossy skin drawn tightly over the joint. No fluctuation was anywhere evident; no apparent thickening of the capsule. The ham-string muscles were very tense, and upon careful examination no motion whatever of the articulation could be detected. The joint was practically anchylosed by the muscular spasm; the tendon of the quadriceps femoris being equally tense with the flexors of the leg, and resisting any attempt at motion. That the joint rigidity was due to muscular action alone, was demonstrated by the administration of an anæsthetic, free motion being then easily developed. The muscular atrophy was also great, and the reflex spasm marked, and excited upon the slightest attempts at motion. Nocturnal pain had been a prominent symptom, accompanied by sudden cries and incoherent mutterings at night. *Diagnosis.*—Osteitis, affecting probably the articular aspect of the epiphysis of the tibia, and *prognosis* unfavorable, including a possible suppuration, a partial arrest of development of the tibia, and anchylosis.

To bring out plainly the symptoms characteristic of these two conditions, I will analyze the important symptoms of each and place them side by side in a differential table:

CHRONIC SYNOVITIS.[1]	CHRONIC ARTICULAR OSTEITIS.[1]
1. Capsule thickened,—Effusion marked.	1. No thickening of capsule evident.—No sense of fluctuation.
2. Natural contour of leg and thigh—Joint outline obliterated.	2. Muscular atrophy marked,—Joint outline clear and distinct,—Joint appears large, on account of the diminished size of both thigh and leg.
3. Motion extensive and nearly normal.	3. Motion *nil.*
4. Resistance to motion *elastic*, and efforts to overcome it not productive of pain.	4. Joint held perfectly rigid by muscular action alone.
5. No reflex muscular spasm present.	5. Reflex muscular spasm affecting both flexors and extensors.
6. No pain present, nor produced by forcible tests.	6. Acute pain upon the slightest attempts at joint motion.
7. No perceptible limp or hesitation in walking.	7. Unable to walk from pain and deformity.
8. Sleep normal,—No reflex osteitic cry.	8. Incoherent cries and "starting pains" occurring during sleep.
9. Femur and tibia in normal relation to each other.	9. Tibia subluxated backward (partial) by muscular action.
10. Symptoms local, so far as those *dependent* on the joint lesion are concerned.	10. General and local neural symptoms directly referable to the joint lesion.

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[1] The author sees no reason to modify this differential table in any important particular. Nor has he seen, or has his attention been called to, any conclusions based upon pathological research which essentially modifies it. [*Note by the author, September, 1923.*]

A further comparison will be unnecessary. Sufficient is here stated to plainly indicate the essential points involved. *Chronic synovitis*—and especially the fungoid variety—is not, in my experience, attended by reflex muscular spasm to any considerable extent. On the other hand, in *chronic articular osteitis* this spasm is an early and progressive symptom, which gradually increases until anchylosis is simulated. My experience also leads me to the conclusion that the initial lesion occurs more frequently in the epiphysis than in the synovial membrane. Either this is especially true of the hip-joint, or the chronic synovitis remains undiscovered until the morbid process, attacking the bone, gives rise to the characteristic symptom of articular osteitis, viz., the reflex muscular spasm. Such a condition of the hip-joint, for instance, as that which affected the knees in the case of Addie D—— would certainly have remained undiscovered many months, for it was not until the effusion was observed that any trouble was recognized by the parents. It is much more probable that the synovial membrane is more frequently affected in the knee-joint (this is certainly true of acute synovitis) and that the epiphysis is oftener attacked at the hip. I do not remember to have observed more than two or three cases where I was able to diagnose a fungous synovitis of the hip-joint. In one of these cases the mother of the patient (James Morrow) did not apply to the dispensary (Dec. 20, 1876) with any thought that a joint disease existed. There was merely a slight and almost imperceptible swelling in the region of the capsule of the hip-joint to which my attention was invited. The history of the boy developed, however, uncertain and vague symptoms of several months' standing, which pointed to the hip-joint—which symptoms had all disappeared upon the appearance of the swelling above referred to. Examination of the articulation developed marked reflex muscular spasm in the extremes of joint motion. The patient could run without a limp. He jumped from a chair without favoring the suspected limb. He could even hop on the affected side with perfect ease. But for the existence of the characteristic reflex spasm and the limited motion (there was no pain, nocturnal or diurnal) I should have thought there was some extra capsular trouble simulating joint inflammation. But this spasm was marked. It was chiefly noted in rotation, both inward and outward, and it possessed all the characteristics of the peculiar resistance which accompanies articular osteitis. After some study, I made a diagnosis of chronic fungoid inflammation of the synovial membrane which had existed for some months, and which was then invading the bone. With this opinion, an extension instrument was applied. The stiffening of the joint became rapidly progressive. This result did not surprise me; on the contrary it served to confirm my diagnosis, for I have frequently observed that in fungous synovitis of the knee-joint, where tolerably good motion existed, that an immobilizing apparatus rapidly stiffens the articulation. I believe this rigidity results from the fixation, and in this way: When the joint is allowed to move freely, the spongy granulations are, by friction and pressure, kept in abeyance; but when the joint remains fixed in any one position for a length of time, they become exuberant and sensitive, and motion is thus interfered with.

After a time (to return to the Morrow case) the rigidity became so marked that the apparatus was wholly dispensed with, in order to test the effect of a free use of the articulation. For a time slightly better motion was observed. Pain then became an urgent symptom. But in order to test the matter fully the re-application of the splint was deferred. The joint daily became weaker; the muscular spasm grew more persistent; nocturnal starting pains appeared, and the patient, on November 1, 1877, was unable to walk a single step alone. The limbs at that time were of equal length, the atrophy marked, and a very slight amount of joint motion only was perceptible. Muscular spasm was very marked, and the faradic reaction of the thigh muscles, which from the first had been modified, showed a still greater reduction. Pain could be developed by very limited force, and all the symptoms of "morbus coxarius" were present. A fact, to which I wish to call attention, was the equal length of the limbs at my last examination of the case (November 10th). The history of the disease covers a period of eighteen months. Had the lesion been one of primary chronic epiphysitis, some shortening would have taken place. The splint was applied for the second time about the 5th of November, since which time the subjective symptoms have been modified, pain being less, and sleep better.

I have said that in the first stage of chronic inflammation involving the joints, but little difficulty occurred in making a differential diagnosis between synovitis and osteitis. My reasons for making this statement are evident in the differential table which I have given, and from my remarks. But many cases present where the articulation is not examined until the disease has existed for a long time. The symptoms here may be conflicting. Evidences of synovial disease may be present, and a decided reflex spasm also exist. My rule under these circumstances is as follows: If the various joint movements, be they limited or nearly normal, are unaccompanied by this peculiar reflex muscular spasm and atrophy, I believe the bone to be free from disease. If, on the contrary, motion be equally extensive, even approximating the extremes, and this spasm occur, the bone is involved. *Marked reflex spasm in chronic joint disease always indicates osteitis.* Its absence, I believe, eliminates the bone, as a rule, from any participation in the lesion. There is a class of cases which form an apparent exception to this rule. I refer to those cases of articular osteitis (either primary or secondary) where early and profuse suppuration occurs. In this condition the reflex muscular atrophy and spasm do not seem proportionate to the extent and character of the lesion. These cases have not occurred very frequently in my experience. They seem to accompany the markedly scrofulous diathesis, and are frequently followed by lardaceous degeneration. I shall speak more concisely of this condition on a future occasion. The opportunities I have had of examining diseased joints after exsection or amputation, confirm my position regarding the relation of the muscular spasm to the lesion. I will mention two or three of the most striking cases, in illustration.

Mr. S—— entered St. Luke's Hospital on Sept. 6, 1876, with chronic disease of the left knee-joint of several years standing. The limb had become, in consequence of the diseased articulation, a source of much annoyance and trouble to the patient, and various efforts had been made, but without success, to afford relief by conservative measures. A very considerable degree of joint motion existed (to exceed 90° of flexion and almost perfect extension). A very perceptible and distinctly audible cartilaginous crepitation existed on passive movement. There was no marked nocturnal pain however, and no decided reflex muscular spasm. It existed, however, slightly. In October, 1876, Dr. Little amputated at the lower third of the thigh. Examination of the joint revealed the characteristic appearance of a fungous degeneration of the synovial membrane in an advanced stage, and the eroded and roughened cartilage had a worm-eaten aspect. The capsule was filled with a pulpy mass of unhealthy granulations, which had invaded the ligaments. The bone was not carefully examined. I should infer that it was slightly diseased, and probably in the head of the tibia. This case is cited to demonstrate that an extensive degeneration of both the synovial membrane and cartilage are compatible with a considerable degree of free and unrestricted motion of the articulation.

Maggie Quinn entered St. Luke's Hospital on Dec. 17, 1875. The patient had sustained an injury of a very uncertain character some six weeks before her admission, which the mother stated had been followed by obscure symptoms about the joint. When examined the motions of the limb were nearly normal, but pain and marked reflex muscular spasm were developed in the extremes of movement, especially when, with the patient in the prone position, and the leg flexed upon the extended thigh, rotation outward or inward was attempted. The child walked with a decided limp, and already a considerable degree of muscular atrophy had occurred. The muscular spasm increased rapidly, and in three months the joint was seemingly anchylosed. The incoherent nocturnal cry occurred soon after her admission. An extension splint was applied about the middle of January following. An incontinence of urine developed, however, which materially interfered with the perineal straps, and excoriations followed which refused to yield to treatment. In June, 1876, my colleague, Dr. T. T. Sabine, excised the hip-joint. The bone and cartilage were found diseased, and the synovial membrane was, so far as could be ascertained, only slightly affected. I do not remember to have seen a more rapidly progressive or a more persistent reflex muscular spasm than was developed in this case. The patholigical specimen obtained sustained my expressed opinion that the primary lesion existed in the bone.

A peculiar circumstance was developed in the after history of this case. Neither the nocturnal osteitic cry nor the reflex spasm of the muscles ceased after the operation. They both existed irregularly, subsequent to the exsection. I forgot to state that the acetabulum was found to be diseased.

This case, operated on so early in its history (a little more than six months after the first appearance of the symptoms), was deemed by me a favorable one for exsection. I regret to add that the sinuses are still unhealed, and that albumin has lately been discovered in the urine.

John Buchanan, aged 3 years, applied at the Orthopædic Dispensary on May 22, 1877, with extensive disease of the right knee-joint. About one year before, his parents had noticed that the patient limped a little—"dragging the limb." No pain was noticed during the day, but at night the patient would "scream out in his sleep." No swelling of the joint was noticed until after the limp and the nocturnal pain. When the limp appeared the joint commenced to stiffen and to gradually flex. The slightest movement at this time produced very severe pain. It was with difficulty that the mother could put on the patient's shoe and stocking. The muscles began to waste, and the "stiffness" of the joint became a marked symptom—progressing until no joint motion was apparent. In December following there was a marked increase of the pain. High fever supervened, and the patient began to lose flesh rapidly. Swelling of the joint now occurred. When he applied at the Dispensary the joint was much enlarged, the tissues surrounding it being thickened and infiltrated. Two sinuses led down to the joint, one above the patella, the other below and to the outer side. An abscess existed on the anterior and outer aspect of the thigh, at about the junction of the middle with the lower third. The muscles of the leg and thigh were greatly atrophied. Any attempt at joint motion produced excruciating pain, and a marked reflex spasm of the muscles existed. The patient was very much reduced in flesh. On May 28th, after consultation with Drs. Markoe, Peters, and Sabine, I amputated just above the abscess, using the abscess wall for the outer flap. The patient recovered rapidly. Examination of the joint revealed the fact that the lesion primarily existed in the head of the tibia, where a cavity was found in the internal tuberosity, involving almost all of that portion of the epiphysis. In the cavity was found a sequestrum as large as a good sized pea. Free communication existed between this cavity and the joint, the cartilage being perforated. Pus had thus found its way into the articulation, producing complete joint disintegration. The condyles of the femur were not involved, a slight erosion only existing on the articular cartilage of the internal condyle. Comment upon this case is, perhaps, unnecessary. I will merely call attention to the initial lesion which was followed by the early stiffening of the joint, the muscular atrophy and spasm, the absence of swelling, and the osteitic cry, all of which existed before the epiphyseal abscess found exit in the cavity of the joint.

In conclusion I wish to call attention to this peculiar muscular spasm in chronic joint disease. It would be a difficult matter to describe it in such a manner that it would be recognised. Like the physical signs in thoracic disease, its peculiar qualities are best acquired by clinical study. I shall attempt to describe it when I speak of the symptoms of joint disease, in detail. I have elsewhere stated that I believe this spasm to be due to an irritation or inflammation of the peripheral nerves supplying the joint, and that the spasm thus induced produces the various progressive deformities in joint disease.[1] Without anticipating what I shall say upon this subject on some future occasion, I may remark that further investigations have only strengthened the position I take in the article referred to. That the trophic changes occurring in joint disease, and the reflex spasm which accompanies it, are due to the same cause I have no doubt. Billroth remarks:

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[1] *Op. cit.*

"The extent to which the muscles sympathize varies greatly; according to my experience the highest grades of muscular atrophy occur in those cases where there is no suppuration of the joints but caries sicca, and where the joint disease seems to proceed from osteitis."[1] In the paper already alluded to I say, "In chronic osteitis, especially if associated with a chondritis, the contraction is firm, tense, and very persistent. It increases with greater or less rapidity, until anchylosis is simulated. The atrophy is slowly but steadily progressive, and the muscles show a marked decrease of faradic reaction. This is especially the case in the dry osteitis." I had not, when I wrote this, seen the above sentence, quoted from Billroth, else I should have referred to it in my paper. The neural symptoms are so marked in some obscure joint lesions as to be suggestive of a primary spinal cord change, a condition which Dr. S. Weir Mitchell calls attention to in his paper on "Spinal Arthropathies."[2] In a letter to me, dated November 5th, he says: "There is a resemblance between the two conditions you speak of," (the muscular spasm and atrophy occurring in joint disease, and that following injuries of nerves), "both sets of contractions and atrophies result from irritative peripheral lesions of nerves, in or out of joints; and in the joints, as in the nerve trunks, the muscular loss is often out of proportion to the joint lesion, and this especially in the knee and shoulder—the small joints scarcely cause it,—but I fancy that more often than we suppose the joint and muscle lesions are common results of a spinal malady." I have often expressed a desire to subject the spinal cord to a microscopical examination, my thought being that in some of these old cases, where caries sicca had existed for years, some change might have occurred in the cord as a result of the prolonged reflex movement. The cases of spinal arthropathy which I have seen, differ in many important respects from the lesion I have attempted to describe, where, as I believe, the neural symptoms are purely reflex.

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[1] *Op. cit.*, p. 481.

[2] *Am. Jour. Med. Sci.*, April, 1876.

THE PATHOLOGY, SYMPTOMS AND DIAGNOSIS OF ANKLE-JOINT DISEASE[1]

A lecture delivered at the New York Orthopædic Dispensary and Hospital, November 3, 1881.

THE ankle-joint, no less than the generally considered, more important articulations at the knee and hip, plays an important part in the mechanism of locomotion. Situated at the distal part of the lower extremity, it forms the basis upon which the whole weight of the body rests during the act of walking, and we may justly admire the mechanism which Nature has provided to render this articulation at once so compact and so strong.

As we shall direct our attention, principally, to the ankle-joint itself—rather than to the foot as a whole—we will, at once, proceed to a consideration of the articulation. This joint belongs to the class Diarthrosis, and to the subdivision Ginglymus. In other words, the ankle is a hinge-joint, and the great accuracy with which the bones are adapted to each other, is one great element of its strength. It is formed by the junction of the distal ends of the tibia and fibula, with the upper and two lateral surfaces of the astragalus. The fibula forms only a minor part of the joint articulating only with the outer side of the astragalus, and its distal end forms the outer malleolus. The tibia, on the contrary, presents a large articulating surface, and its prominent process forms the internal malleolus. These two bones then form a sort of archway, irregular in shape. They are bound together by ligaments, and this union is so strong that, as is well-known, a fracture of the bones themselves is much more frequent than a laceration of the ligaments, which hold them in position. Into this irregular arch, formed by the two malleoli, the astragalus fits with a degree of accuracy which is surprising. And when the foot is placed upon the ground in walking, the astragalus presents a surface over which the tibia and fibula glide, and each astragalus, in turn, bears the entire weight of the body. In conditions where these apposed surfaces are inflamed, this gliding motion is interfered with, and the structure of the tarsal bones and tarsal ligaments does not permit any vicarious action. Indeed, the limp of ankle-joint disease is characteristic, so also is the limp of knee-joint disease, and the "hip limp" is easily recognized. An expert in orthopædy can almost always make a pretty accurate estimate of the location of the lesion before his patient is undressed by the peculiarities of attitude, gait, etc., and these remarks are especially true of certain forms of ankle-joint disease.

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[1] *Annals of Anatomy and Surgery*, January, 1882.

The astragalus articulates with the os calcis and the scaphoid. If disease of either of these bones actually involves the astragalus, we find some of the symptoms of ankle-joint disease. But in tarsal disease, *per se*, we see an altogether different train of symptoms. I will not stop here, however, to describe these differences. We will consider them under their proper head.

It is well that we should notice a few anatomical points —as they will have a practical bearing upon some of the questions we shall discuss later. There are four ligaments: the anterior, the posterior, the internal and external lateral. They form together a complete envelope of the joint, and in some instances pass beyond and assist in forming the capsule of some of the tarsal joints. It is stated by some anatomists, and with reason, that the synovial membrane of the ankle-joint is very extensive. It lines all the ligaments just mentioned, and sends a cul-de-sac to the inferior articulation between the tibia and fibula. This synovial sac is supposed to contain a larger quantity of synovia than that of any other in the body. It would certainly seem otherwise, especially when we compare a simple synovitis of the ankle with the same condition at the knee. But it must be remembered that in the former articulation the synovial membrane is held down by these strong ligaments, while in the latter an effusion easily distends the soft, fibrous membrane which covers the easily distensible synovial membrane. In the ankle-joint effusion in the synovial sac shows itself principally along the anterior portion of the joint, and under each malleolus. But little impression is made posteriorly, and it has rarely occurred, in my own experience, in a case of uncomplicated synovitis of this articulation, that the fossæ on either side of the tendo Achillis became obliterated. Not unfrequently in extensive disease which involves the whole articulation, these fossæ become lost through an œdematous infiltration. But these cases do not involve the synovial membrane alone. The ligamentous protection is so strong and so complete that nature affords the most excellent means of producing compression. It is not until considerable deposit or distention has occurred in ankle-joint synovial disease that the characteristic bulging is very perceptible. This, with other factors, to be mentioned later, is a reason why chronic fungoid synovitis is not sooner recognized.

It is well that we should stop a moment and consider carefully the bursæ about this articulation. Their number is considerable, and their location is such that it might be very easy to mistake a simple chronic bursitis for a chronic synovitis. I have seen cases where this mistake was made; and when we consider that these bursæ may become quite large from injury or during a process of chronic inflammation, it is not unlikely that the same error will occur again. I have seen a case where two or three contiguous bursæ over the anterior part of the joint became simultaneously affected from injury, and the imitation here was very close, for there was considerable swelling about the entire joint also. There is one bursa each for the tibialis anticus, the extensor longus pollicis, and a third one common to the extensor longus digitorum and peroneus brevis. Behind the external malleolus there is a bursa common to both peroneus longus and brevis. It continues downward on the peroneus longus as far as the sole of the foot. But there is still another in front of the external malleolus; this is the bursa proper to the peroneus brevis. There is another for the flexor longus pollicis. This extends from some distance above the lower end of the tibia to beyond the groove in the sustentaculum tali. There are bursæ also which assist the movement of the muscles over the lower end of the tibia, over the internal lateral ligament; also one over the tibia for the tibialis posticus tendon.

We can readily see, therefore, how a chronic bursitis in certain locations may resemble a synovial effusion or synovial degeneration without effusion, just as we can imagine a suppurating bursitis in the neighborhood of the knee-joint may be called "white swelling." A close study of the symptoms, however, and a careful consideration of the history should enable you to decide in all questionable cases.

All the muscles which *arise* from the tibia and fibula pass *over* the ankle-joint. Two which arise from the condyles of the femur are inserted beyond the ankle. These are the gastrocnemius and plantaris. The former group act directly or indirectly upon the ankle-joint, while the latter possess the power of flexing the leg and extending the foot. Thus, while the peronei, for example, may produce by their contraction a position of valgus, the gastrocnemius may be considerably shortened without apparently causing any equinus if the knee be flexed. So that, in conducting your examinations of the muscular elements of an ankle-joint deformity, whether the case is one of ankle-joint disease or of club-foot, be sure the knee is fully extended. In this way only can the amount of posterior muscular resistance be estimated.

The inner border of the foot is raised and adducted by the tibialis anticus and posticus; the outer border of the foot is raised and abducted by the three peronei. The tibialis anticus and peroneus tertius, acting conjointly, flex the ankle-joint, and the peroneus longus and brevis and tibialis posticus extend the foot. The arch of the foot, though held in position by strong ligaments, is materially supported by the tibialis anticus and the peroneus longus. The flexor longus digitorum and flexor longus pollicis flex the toes to which they are attached, and when this action is completed they assist in extending the ankle. The other extensors and flexors also materially assist the larger muscles in flexing or extending the ankle-joint, though, of course, the calf muscles are the powerful extensors of the articulation.

The fact that the foot is abducted or adducted by certain muscles, has been noted. It must not be thought, however, that these movements take place at the ankle-joint. Being a true hinge-joint, no movement takes place at this articulation but flexion and extension—unless it be placed in a position of extreme extension, and then there is a slight lateral motion. During flexion and extension the foot does not move through a direct antero-posterior plane. The plane upon which motion does occur is "inclined outward and drawn through the foot from the middle of the astragalus to the apposed surfaces of the bases of the second and third metatarsal bones," and consequently there is a slight movement outwards of the foot in flexion, and inwards in extension.

We have thus briefly noticed a few points in the anatomy and mechanism of the ankle-joint (some of which have been taken from Morris's excellent work on the "Anatomy of the Joints") for the reason that in by far the greater number of cases, clinically considered, contraction of some or all of these muscles occur, and the resulting deformity, which by many has been referred directly to changes which take place in the joint itself, is due to disturbances of muscular action. We shall especially consider these conditions later.

The diseases of the ankle-joint which will especially engage our attention to-day are those which can only be described as chronic, and which would be ordinarily grouped under the head of *tumor albus* or white swelling. And they may be described as presenting, at least clinically, a primary lesion of either the synovial membrane or the bone. If chronic synovitis, or osteitis of the ankle-joint, ever has its inception in a primary non-traumatic lesion of the ligaments or cartilage, we have no absolute demonstration of the fact, and from what we know of their structure and functions we may be assured that an idiopathic chronic inflammation of these structures is very rare. The cartilage, especially, in a state of chronic degeneration, would give no sign, and the fibrous structure of the ligaments leads us to look for acute rather than chronic lesions. And, clinically speaking, these conclusions hold good.

A great deal has been written and said concerning the ætiology of chronic joint disease. We have but one opinion upon the subject, and this opinion has been strengthened by daily clinical work in this class of cases, and a study of the pathological conditions discovered. If we accept the story told us by the ankle-joint, we are only strengthened in our view that traumatism plays a very unimportant part in the production of chronic disease. The ankle is peculiarly exposed to violent shocks and twists. It is in almost constant use during the day, and its location is such that great and often unexpected shocks are thrown upon it. We have many sprains of the ankle, and but few, very few, dislocations. And although the sprain may be very serious, apparently, and ecchymotic areas may be found quite extensive in character, it is very exceptional that we find a chronic joint disease of the fungoid type following such injuries. Almost always some injury is blamed; almost always the relation of cause and effect is associated in the minds of the laity between a slight twist, not followed by any especial trouble, and the retrograde lesion found in chronic joint disease. Eminent authorities take this view also. We have failed to be convinced by their arguments and can only say, as we have expressed ourselves on former occasions, that we deem the development of a typical, chronic fungoid joint lesion, in a perfectly healthy person, almost impossible. We cannot trace the regressive metamorphosis which characterize these conditions—the degenerate abscesses, the flocculent, ichorous discharge, the prolonged dry caries or synovial degeneration—to traumatism alone. It requires a peculiar predisposition, I firmly believe, to make traumatism avoidable as a factor.

Clinically, then, as well as pathologically, we divide the chronic diseases of the ankle into two classes—those which affect the synovial membrane alone, and those which are primarily developed in the bone. Here, as in the knee-joint, the study of joint lesions, clinically speaking, is quite easily pursued, for these joints are superficial, and we can arrange our aids to diagnosis into objective signs and subjective symptoms. Chronic synovitis of the ankle-joint *per se* may exist for a long time without giving any subjective symptoms whatever. Uncomplicated chronic osteitis presents subjective elements of diagnosis almost wholly. It should not be difficult, therefore, to differentiate between the two; and as the treatment to be pursued is wholly different in these two conditions, it is necessary to reach positive conclusions.

You may ask: What are the changes which take place in chronic fungoid synovitis? I answer, briefly, that in the large number of cases the primary changes occur upon the free surface of the membrane, and they are characterized by what the German pathologists call fungous or granular degeneration. I have not time to stop here to enter into a minute detail regarding these changes. They are fully described by Billroth, Volkmann, and other writers, and I would advise you to familiarize yourselves with their descriptions. Suffice it to say, that sooner or later this fungous process produces a modification of the synovial fluid. The synovial membrane becomes distended or simply thickened by the gelatinous deposit, but there is no deformity save that which results from the thickened membrane or the distention of the capsule. The membrane presents very few nerves, and they are not highly sensitive. Hence, pain is not complained of, and locomotion is practically uninterfered with. Joint motion remains nearly normal, and unless the swelling of the articulation is noticed and successfully treated, the joint lesion progresses slowly, but none the less surely. But, after a while, pain, limping and real debility of the limb supervene. The symptoms become more urgent, the joint shows a higher temperature, the muscles begin to waste, sleep is disturbed, and the joint becomes almost useless. What do these new symptoms mean? The fungous, proliferating masses have penetrated the nerveless cartilage, and are, as Billroth expresses it, eating their way into the bone. This fungous proliferation attacks the fibrous structure of the bone. It attacks it primarily, leaving the very sensitive nerve filaments exposed to these fungous masses and the bony detritus. The muscular spasm, the muscular atrophy, the pain, the debility of the limb, the nocturnal cry—all these occur in fungous caries, and, in some cases, abscess occurs. In other cases the disease goes on for years, and yet no abscess appears. In many of these cases we have a condition of caries sicca—a condition described with considerable minuteness by the German pathologists, and one which particularly demands our attention; for these cases especially, where the subjective element so strongly preponderates without the objective signs, are sometimes called hysterical, or the limp is ascribed to habit, etc. The objective signs of joint disease which are so depended upon by many general surgical writers, are wholly wanting. Be on your guard, therefore, in studying joint disease. Apparently easy of diagnosis, these chronic joint lesions have deceived the best surgical talent, and a close study of all the symptoms, and a very deliberate analysis of all the elements of diagnosis—including all the methods of precision applicable to this class of cases—is necessary to the correct understanding of the conditions met with in even chronic disease of the ankle-joint. Be careful in obtaining the history; omit no point that gives you a clue; follow up every suggestion which any symptom presents; and, in many instances, you will, to say the least, be surprised at the clinical picture which will result.

In making a diagnosis of chronic ankle-joint lesions, you will consider—(1.) Chronic synovitis, and this term we use in the sense of chronic fungoid synovitis unless otherwise specified; (2.) Chronic osteitis, and under this term we will include to-day the various forms of osseous inflammation of the regressive type, whether dry or humid; (3.) Extra capsular, or peri-articular lesions which simulate real joint disease. This classification will be found to answer all practical and clinical purposes. On some subsequent occasion I shall enter more into the detail of the pathology of joint lesions. I wish to-day to impress upon your minds the important clinical aspects of the two great classes, chronic synovitis and chronic osteitis.

Chronic synovial inflammation gives very few symptoms, oftentimes none at all in the early stage. Its onset is very insidious, and it progresses very slowly; months being necessary, in many instances, to enable the parent of the child to determine whether or not there is any trouble; and the fact that the child does not complain of pain insures a procrastination in the greater number of instances. But when the disease has sufficiently advanced to produce subjective symptoms, when the swelling is very perceptible, when the patient commences to limp after a long walk, and there can be no error in the conclusion that there is something serious, the surgeon is consulted. It not unfrequently happens that it is then too late to secure the best result.

I have seen many cases of what I have called uncomplicated chronic synovial inflammation, of the variety described under remarks on pathology—viz., the fungous, proliferating degeneration, which may consume much synovial tissue without producing any pain whatever. In these cases there is no pain, but the bulging and thickened synovial sac contains either an altered secretion or a mass of fungous material. I know this, because I have frequently seen it, and the microscope proves it. But there is little or no true pus. And this emphasizes the conclusion that the morbid changes which take place in chronic joint disease are not inflammatory changes as usually understood. To return to the symptoms. There is little if any limitation of joint movement. Flexion and extension are normal, or so nearly so that careful tests alone can demonstrate any modification of normal movement. The patient bears quite rough handling of the articulation without complaint. Concussion does not produce pain or flinching; extra movement gives little or no pain. There is usually no cartilaginous crepitation, no involuntary muscular resistance, and no muscular wasting except that due to disuse. The gastrocnemius, the lateral and the anterior muscles, show no material reduction to the faradic current. It is a chronic degenerative lesion of a simple serous membrane, uncomplicated by any extensive change in the cartilage, and unaccompanied by any osseous inflammation. Such cases as these fall to the notice of every hospital surgeon, and in some instances their true nature is not recognized until a considerable deposit of fungoid or granular matter has occurred. But some of these cases have a subsequent history. They may progress in spite of any treatment which is adopted. These fungous, proliferating masses, in certain instances, go on in their work of destruction: they invade the cartilage. It succumbs to their progressive movement, especially if the traumatism of locomotion is not prevented; but still there is no pain, so long as the lesion is confined to the superficial or articular surface, although the debility of the limb is increased. But finally the work of disintegration becomes almost complete. The surface of bone lying under the cartilage becomes affected, and these granular, fungoid masses find new fields for labor. With an energy which is unmistakable they attack the cancellous structure of the bone, and the minute nerve filaments which are so abundant in all epiphyses begin to show the effect of exposure to this peculiar morbid process. It is now that the limping amounts to a real debility, and that pain, muscular spasm, and muscular atrophy appear. The muscles do not so readily respond to the faradic current. The patient becomes more or less disabled, and crutches or some artificial aid to locomotion is introduced. If not treated at all, they finally get well with deformed and stiff joints, for it is very unusual for children with ankle-joint diseases to die from the disease under consideration. We have no record of any such result. Here, then, you have a brief clinical description of chronic synovitis, which finally also becomes a chronic osteitis of the ankle-joint. The symptoms are objective in the first part of the history, both objective and subjective in the second part. There is a peculiar lesion of an almost nerveless tissue at first, followed by a progressive and destructive extension of this lesion through the cartilage to the sensitive and vascular epiphysis. There should be no question about your diagnosis in these cases. Chronic synovitis and chronic osteitis of the ankle-joint should never be confounded—though, of course, they frequently co-exist—when, also as a matter of course, the osseous symptoms are those which are important and urgent.

The progress of this disease may be the reverse—that is, you may find symptoms of bone disease with no effusion or deposit in the synovial membrane. I have seen many cases where there was no evidence whatever of synovial disease. The initial lesion commenced in the bone, and did not involve the surrounding structures, for the fungous caries, the granular caries, the dry caries, or the silent caries (whichever you may see fit to call it), frequently exists for years uncomplicated by synovial disease; or if this membrane does become involved, it is late in the history of the lesion and after the cartilage has been destroyed. Under these circumstances we find the pronounced limp first, then the spasm and atrophy, without any evidence whatever of synovial disease or synovial degeneration.

The symptoms then to be noted are: In synovial disease, (1) change in the outline of the joint, and (2) a sense of fluctuation, or one which resembles it; a doughy, elastic feeling. Accompanying this state there is an almost normal use of the joint without any pain or marked limping, the latter occurring after long or fatiguing walks. In chronic disease of the bone we find—(1) an involuntary reflex muscular spasm which may render the joint so stiff as to suggest anchylosis; (2) pain, either orally expressed or manifested by an involuntary and very suggestive shrinking and facial expressions; (3) an absolute inability to use the joint in walking in many instances; (4) muscular wasting not accounted for by simple disuse; (5) reduction of the faradic contractility of the muscles affected by the reflex muscular spasm. In ankle-joint disease the nocturnal osteitic cry, which is very often noted in disease of the hip, knee, and spine, is not frequently present. It does occur sometimes, however, and should always be sought for. In uncomplicated synovial disease these purely involuntary and reflex symptoms do not appear. The clinical difference between these two conditions is plain and unmistakable, as I shall hope to show you.

Chronic bursitis may be mistaken for chronic synovitis. But the careful observer will not mistake it for chronic osteitis, for reasons which you can now appreciate. Bursitis may deform the region in which you would expect to find synovial effusion, and objectively give all the signs of synovial disease or distension. But if you are in doubt, use your hypodermic syringe and draw a quantity of fluid from the sac. The effusion of chronic bursitis is, practically speaking, an increased or slightly modified normal secretion; the contents of the synovial sac in chronic degenerative synovitis are far different, and in some instances you will not be able to obtain any fluid whatever.

Tarsal disease may arise and closely simulate ankle-joint disease, or by involving those articulations to which the synovial membrane of the ankle-joint sends reflections, may lead to secondary and true inflammation of the ankle-joint itself. But also bear in mind that we draw a distinct line between chronic synovitis and hydrarthrus. The former, we hold, means a regressive metamorphosis of joint tissue; the latter signifies an increased natural or nearly natural secretion. In considering tarsal disease, therefore, bear in mind this distinction, as well as in other conditions. Note also that in tarsal disease you find almost always *bone* disease, not an inflammation of the inter-tarsal synovial membranes, which undoubtedly occurs; but as at the hip-joint synovial disease is not easily recognized. And the important element of reflex muscular spasm helps us greatly. It is a rule to which there are few, if any exceptions, that *true* reflex muscular spasm is found only at the diseased articulation: for example, in hip disease we have reflex spasm, limiting motion at the hip, but *not* at the knee, though we have muscles which act on both articulations. So in tarsal disease: for example, again; medio-tarsal disease, *per se*, does not produce true reflex spasm *at the ankle.* I have seen extensive tarsal disease without any interference with ankle-joint motion. And you may know that you have no extensive *bone* lesion at the ankle-joint if you find the movements normal or nearly so; and reflex muscular spasm does not enter into a consideration of tarsal disease any more than it would into a consideration of sacro-iliac disease, for instance. If there should exist astragulo-scaphoid, or calcaneo-astragular disease, remember what I have said already—viz., that so long as the true articular surfaces are free you will not find the symptoms which characterize ankle-joint osteitis.

You may find neuromimesis (a term introduced by Paget to indicate hysterical affections) of the ankle-joint. Even in children you may find this condition. Neuromimesis of the ankle-joint may simulate either chronic synovitis or chronic osteitis. The joint may be swelled, hot, painful, and very stiff. You may have apparently all the important signs of chronic or acute joint inflammation, but if you have once mastered the clinical aspects of reflex muscular spasm you will not be deceived by these cases, and the differential diagnosis between the hysterical ankle and chronic synovitis cannot be difficult if you are acquainted with the natural history and course of the true disease. A close study will develop some important variation not consistent with the true disease. But I will mention one or two points that may aid you. The conditions in true disease do not vary. Though subject to periods of remission in its first stage, the tendency of ankle-joint disease, whether synovial or osseous, is to slowly and surely progress. The neuromimetic varies greatly and without regard to any recognized law. On the other hand this granular caries, this fungoid caries, this silent caries, is very suggestive of simple functional disturbance. Esmarch was in doubt regarding such a case as this, and the foot was finally amputated for extensive disease. I have seen many cases of real joint and spinal disease going slowly but surely to destruction with a diagnosis of hysteria, malaria, or neurasthenia, when this silent fungoid disintegration was going on. Don't fall into the error of calling real disease *hysterical*. If you err at all, give your patient the benefit of the doubt. You had much better unnecessarily treat a hysterical joint, than to aggravate the morbid state, by adding the element of traumatism, which is destruction itself.

The position assumed by the foot in chronic osteitis of the ankle-joint is due not to any intra-articular effusion, nor, ordinarily, to any loss of substance in the joint ends, but to muscular spasm. The fact that the inter-articular effusion has nothing to do with this deformity is proved by the fact that the synovial membrane may be distended very greatly, as in hydrarthrus, and yet no malposition occurs, while in that form of disease which is characteristic of osteitis, and where there is no effusion at all, the malposition is the most exaggerated and the most difficult to overcome. This muscular spasm exists in chronic osteitis always, especially when the articular lamella and cartilage are involved; it is never present in uncomplicated chronic synovitis, and its presence is, while a conservative effort on the part of nature, a constant source of irritation and danger to the joint. It is always present when the stimulus of over movement is applied, and no effort of the patient's will-power can successfully combat it. Sleep does not modify it; opium or chloral cannot, in ordinary doses, annul it. But one thing within the range of known therapeutical agents that I have tried can overcome it, and that is the profound anæsthesia of ether or chloroform. Bear these facts in mind, they will be useful to you; for I have known eminent authorities to err in diagnosis because the suspected joint moved easily under anæsthesia.

The foot, in ankle-joint disease, may assume almost any position. It rarely presents as a varus deformity, however; nor does the joint always become extended, as you would naturally expect, for the extensors are the strong muscles at the ankle, and it is a rule at other joints that the limb takes the direction of the stronger group. You can easily imagine that the patient would present the heel to the floor and bear his weight fairly on the affected articulation, rather than to extend the foot and thus add motion to pressure in locomotion—a condition always avoided by patients with chronic articular osteitis. But the foot is, almost always, nearly straight, or there is a more or less prominent valgus, with slight flexion or slight extension. And if you remember what was stated when we spoke of the normal motion of the ankle, and that there was a movement outward of the foot when flexion occurred, you will see why the valgus occurs with flexion.

The more attention one devotes to chronic ankle- and knee-joint diseases, where the morbid processes may be closely watched, from a clinical standpoint, the more one will become convinced that the ground taken by us regarding the relation of muscular atrophy and muscular spasm to chronic joint disease is correct. Their value, in a diagnostic point of view, is great. Indeed, these two, muscular atrophy, and even bone atrophy and reflex muscular spasm, form the basis upon which a correct diagnosis is to be made in certain instances—just as their absence is of value. I therefore call your especial attention to them, and shall ask you all to follow me closely in my tests, and to avail yourselves of the clinical opportunities which will be afforded you here to study them.

In order, therefore, to fully prepare you for your study I will close by speaking of the most important of these diagnostic elements, for they are as applicable to knee and hip disease, which we shall consider later, as they are to ankle disease, which we are considering to-day.

Reflex spasm, as presented in chronic joint disease, may be defined as an *involuntary* muscular protection of an inflamed articular surface. Bear in mind the qualification *involuntary*. You will soon learn, by clinical experience, the great difference between a voluntary, and hence irregular or varying muscular resistance, and the involuntary and hence, in this instance, unvarying muscular spasm. Of the cause of this involuntary spasm I shall speak later. I wish now to particularly emphasize its clinical features. The muscles which resist are not generally in a state of permanent spasm. It is only when anchylosis is simulated that the spasm is always present. If you flex and extend an inflamed ankle-joint with osteitis, you find in many cases a very considerable degree of movement. But before the normal limit is reached the muscles which oppose become suddenly *tetanized*. The very instant the inflamed surface or surfaces becomes unduly pressed upon there is an involuntary muscular resistance, and the resistance remains until the pressure is removed. You cannot overcome this spasm except by very severe measures or the administration of an anæsthetic, and the patient shows in many ways the apprehension and suffering which a persistent force entails, and an aggravation of the symptoms always follows free motion under anæsthesia. This spasm is nature's protection, and she uses it well. You cannot fail to detect it when once you have learned its peculiar properties, and once having mastered the art of recognizing it you will regard it as *the* important diagnostic sign.

Much the same may be said of reflex muscular atrophy. As I have pointed out to you already, in chronic *synovitis*, *per se*, you do not find either reflex spasm or reflex atrophy. In chronic osteitis you find both these elements to a greater or less degree, and they both depend upon the same cause.

When the nutrition of an epiphysis is interfered with you find bone atrophy, and synovial inflammation certainly does not interfere with epiphyseal nutrition. Not unfrequently in ankle-joint osteitis I have found the tibia from one-quarter to one-half inch shorter than in the normal, as shown by the opposite or healthy one in children of five or six years. And so regarding the muscles in chronic osteitis. There is a direct relation between the location of the inflammation and the amount and the character of the muscular spasm, the muscular atrophy and the bone atrophy. If the astragalus alone is diseased we get foot atrophy, more particularly, though also you find calf wasting, but, not likely, a shortened tibia or fibula; and in some cases of ankle-joint osteitis you find that the tibia alone is shortened. All these things should be considered as a matter not only of diagnosis, but also as a matter of treatment. Let me impress you finally with the great necessity of careful study in all your cases. The field of orthopædy is not fully explored, as yet, by any means. There are many brilliant gems awaiting the patient investigator.

There are many other matters which I would like to bring to your notice to-day. But time does not permit, and they are as applicable to knee and hip disease as to the conditions we have attempted to present this afternoon. I will, therefore, call your attention to them later in the course, and will occupy the remainder of the time at our disposal by showing you typical cases of chronic synovial and chronic osteitic disease of the ankle-joint. We shall take up the subject of prognosis and treatment at our next meeting.

THE PROGNOSIS AND TREATMENT OF ANKLE-JOINT DISEASE[1]

A lecture delivered at the New York Orthopædic Dispensary and Hospital, November 10, 1881

In a previous lecture[2] I called your attention to two important conditions of the ankle-joint—viz.: chronic synovitis and chronic osteitis. Before beginning the consideration of treatment I will briefly call your attention to the prognosis of these lesions.

All joint diseases, and especially those under consideration, require the element of time in their treatment. They are well calculated to test the patience, the industry, the ingenuity, and the temper of the surgeon. In many instances the symptoms will be so unimportant in the minds of the parents, if the patient be a child, or in the consideration of the patient himself, if he be an adult, that complete relief will be looked for in a very short time. But do not let the apparently unimportant symptoms mislead you. You may make it a rule to give an unfavorable prognosis in point of time, in certain chronic joint conditions, if the initial symptoms are apparently unimportant. In other words, the nearer the disease approximates an acute inflammation the better is the prognosis. The more insidious the onset, the longer, as a rule, the disease will continue, and the more uncertain are we regarding the duration, the course, and the ultimate result. Do not attempt, therefore, to give a prognosis until you have duly weighed each symptom, and carefully studied each phase and feature of the history. I have sometimes been tempted to class chronic joint disease as one of the self-limiting lesions. Many of its clinical and pathological aspects lead to the conclusions so ably stated by Dr. Flint regarding phthisis.[1] In other words, all that we can do in certain cases of chronic joint-disease is to watch, support and protect just as we do in certain acute diseases; only in many acute lesions the course of the disease is known, and in chronic joint lesions it is not known. Hence, I am very cautious in giving a prognosis in any chronic joint trouble. It is too much like guess-work; and he who guesses in orthopædic surgery will come to grief. It is my plan, to speak frankly to the patient or his friends, and to state plainly both the character of the lesion and the disadvantages under which the surgeon labors. My prognosis, therefore, is rather a description of the nature of the treatment and of the care necessary to obtain a good result. I do not believe that anyone is justified in making an exact prognosis in chronic joint disease. I do believe, however, that, unless the case is a very bad one, a good result may be assured in childhood; and this is especially true of ankle-joint disease. But you must appreciate the effort necessary to secure this good result. I cannot speak any more definitely than this. Nor can I speak with any greater certainty until there is some more satisfactory method devised for dealing with the known pathological conditions to which I have called your attention. When we can successfully transform a chronic regressive metamorphosis into an acute reparative lesion we can make a much more favorable prognosis. But we cannot accomplish this transformation in all cases; indeed, with our present knowledge, we can succeed in only a very few.

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[1] *Annals of Anatomy and Surgery*, May, 1882.

[2] "The Pathology, Symptoms and Diagnosis of Ankle-Joint Disease," *Annals of Anatomy and Surgery*, vol. v., p. 1, January, 1882.

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[1] See "Self-Limitation in Cases of Phthisis," by Austin Flint, M.D., *Archives of Medicine*, vol. i., No. 3.

Some surgeons seem to be afraid of producing anchylosis by immobilizing a joint in a state of chronic inflammation. I wish we could accomplish this end by so simple a procedure; but anchylosis, in chronic arthritis, is not so easily produced. You may stiffen a joint more or less in a state of chronic inflammation, by an immobilizing apparatus, but you will very rarely produce anchylosis by this means. The disease has progressed, and it will progress in certain cases, notwithstanding every effort we may make. It would be a great victory for orthopædic surgery, could some procedure be devised, short of excision of the diseased articular ends, which would produce anchylosis or synostosis when necessary. In the acute joint lesions it is a very different matter. I have seen them rapidly anchylose under immobilization. And I may say that, as a rule, the effort of the surgeon in certain acute joint lesions is to *prevent* anchylosis. On the other hand, in certain forms of chronic articular disease, it is almost impossible to produce this result. In the latter case the condition does not attain a point where repair takes place until, in many instances, the epiphyseal structures are destroyed. The conditions met with are so perplexing and so little understood, and the indications are, apparently, so contradictory in some cases that it is not a matter of surprise that almost every surgeon should deem his own method of treatment the best, when, in reality, all are more or less faulty. We will try to-day to meet the indications as nearly as may be, simply saying that no treatment yet devised meets all the indications that the pathological conditions present.

If we find a distended capsule at the ankle-joint filled with fungous proliferating masses, the first question to be answered is: Does the disease involve the bone? For we should at once recognize the fact that however painless the lesion may be, however slight the inconvenience it occasions, the process is inherently destructive and progressive. Should we decide that the bone is free from disease or only slightly involved, we must at first make every effort to prevent a further progress of the proliferating fungoid masses.

Bear in mind also, that there are various pathological states in that condition which, for brevity and convenience, I have classed under the general heading of chronic osteitis. Those most frequently met with are:

1. OSTEITIS FUNGOSA OR GRANULOSA, either primary, i.e., occurring in the epiphyseal structures, or secondary, as a sequence of a fungous degeneration of the synovial tissue.

2. CARIES NECROTICA, a condition in which the process of destruction is rapid, and carious bone is discharged, sometimes in quite large pieces.

3. SIMPLE SUPERFICIAL CARIES, following periostitis, and finally—

4. CARIES INTERNA CASEOSA. The prognosis varies somewhat in all these conditions, as well as the treatment. I have spoken of the prognosis in caries fungosa, and I stop again to impress upon your minds that it is a very sluggish condition, with very insidious prodromata. It is accompanied sometimes by the atonic form of suppuration. You do not get laudable pus in this form of abscess. The discharge is profuse, but thin and ichorous and filled with broken down tissue. In the necrotic caries the prognosis is more favorable; nature rapidly exfoliates the diseased bone, and the process is more like that which occurs in necrosis of the diaphyses of the long bones. In

these cases, too, you will find more marked constitutional disturbance. The process, in other words, more nearly approaches the acute form of disease, and it runs a much shorter course, recovery, with anchylosis, in many instances, taking place. The superficial caries, properly recognized and treated, may also be called a favorable lesion, for it may not involve the articular surfaces. It may, however, become the caries necrotica and total joint destruction may ensue. Suppuration of the more favorable type frequently accompanies this process, and the rule should be to open as early as possible, contrary to my usual custom. The caries interna caseosa, is a form very difficult to recognize, especially in the early stage, and it is very apt, with caries fungosa, to be called tuberculosis of the joint. I do not believe, however, that it is possible to differentiate, clinically, between these two conditions. These two, the fungous or dry caries and the caseous caries, are forms of disease that will principally engage our attention to-day. The other forms of disease are those which are more readily recognized and more easily treated, and I will not attempt to discuss them in detail to-day. Nor can I attempt to describe all the chronic joint conditions which we may meet with. Neither can I discuss the various theories regarding their tubercular or non-tubercular origin. I shall rather strive to impress some facts upon your mind regarding the most neglected and I think the least understood of these lesions.

But there are these forms of chronic osteitis of the joints to which I have so briefly alluded; and, while in many instances the differential diagnosis is very difficult, especially in the early stage, you may be assured that the treatment to which I am about to call your attention will answer an excellent purpose in all the pathological states mentioned, whether you supplement your mechanical and systemic aids by incision, excision, etc., or merely pursue the method known as the "expectant plan."

There are many points concerning the etiology, pathology, symptoms and prognosis of chronic joint disease, that I should like to call to your attention before proceeding to the consideration of the treatment of ankle-joint disease; but it would be impossible to do the subject justice in one or even several lectures. We have yet to consider knee, hip and spinal diseases, before we close the discussion of these subjects, and you must pardon me if I defer some questions, and more especially those which are particularly suggested by other joint lesions, to a later date.

I showed you, upon the occasion of my previous lecture, two cases of ankle-joint disease. One of them illustrated the clinical aspects of chronic synovitis, the other of chronic osteitis. In the former we found the joint motions nearly normal; a bulging of the joint capsule, an elastic feeling very suggestive of fluctuation, and an unimpaired walk and attitude. The other presented very different symptoms. The joint motions were very materially impaired by a very pronounced reflex muscular spasm; indeed, anchylosis was simulated by this peculiar condition. The joint outline was not obliterated, and there was no synovial bulging. The patient walked on his heel and limped very markedly; there was also a history of disturbed sleep. A difference in the condition of the muscles was also found in these two cases. The patients with chronic synovitis presented a well-rounded calf, matching its fellow, while the one with chronic osteitis showed a smaller calf when compared with its mate. The former had neither reflex muscular spasm, nor a localized muscular atrophy, the latter illustrated both of these conditions. I cite these cases again, and once more call your attention to them; because we shall make them the basis of our remarks upon treatment, and shall have made for them apparatus to be applied in your presence during the present course.

But, before we speak of mechanical treatment, let us stop for a moment and discuss constitutional treatment.

There are certain indications which must be plain to all who have studied these cases, but it is very important that we should recognize that we have a regressive lesion—a retrograde rather than a reparative process—to deal with. We will suppose it to be the fungous synovitis or caries, and in all cases, not otherwise specified, we will refer to these conditions in our remarks. What remedy or remedies are there which will act specifically upon this fungoid degeneration. We must answer frankly that there are none. Our very ignorance upon this subject drives us at once to the ground of all truly conservative surgeons—viz.: that of thorough, systemic treatment. We must make every effort to restore to nature its impaired reparative power; to bring to the local expression of a peculiar and little understood diathesis a greater plastic effort. But here again we are met by a state of affairs that requires much thought and study. Hygiene, exercise, diet, and many other things are to be thought of; but I know of no specific that I can recommend to you as applicable to all cases, or even one class of cases. Each case is a study, from the hereditary tendencies to the local condition of the joint, and it will require much thought, much study, much experience and observation to apply the remedies best adapted to a case of chronic joint disease. But do not neglect constitutional treatment, though you find that your pet remedy does not produce the impression it should; and, on the other hand, do not depend upon local or mechanical treatment alone.

Some cases are so very discouraging that you may almost be forced to admit, as I have done, that but little could be accomplished by constitutional aid, and that the disease was one which would run its course in spite of all treatment, and get well with a certain amount of joint destruction when the period of limitation was reached, or die in the effort. For in some instances, in spite of all you can do, the apparently simple chronic synovitis will progress and become, in addition, a chronic osteitis. Suppuration of the atonic form may follow, and lardaceous degeneration or tubercular meningitis may remove your patient. These very rarely follow in ankle-joint disease; the hip and spine are most likely to be complicated by these conditions. But one should always take them into account. All this may be very discouraging, I know, but we can only learn by looking at things as they are, not by discussing them from a fictitious standpoint. I have seen chronic joint disease recover with remarkable rapidity, so much so that in some cases I have doubted the correctness of my own diagnosis; but I know that we can, in the majority of instances, help nature very much, and by simply removing traumatism and by aiding nature, as indicated, we can secure the most excellent results, and render our patients, while under treatment, active and happy members of society—a condition of affairs not attained by those who ignore the benefits and curative influence of scientifically constructed apparatus.

The secret of successfully treating chronic joint disease, however, lies in its early recognition. Learn, therefore, to recognize these insidious lesions before great damage is done; not only that, teach the mothers and nurses, that they may know, that many of the slight limps attributed to a tight shoe, or to habit, etc., mean something serious. Teach your families that many cases of "worms" and "indigestion," etc., have turned out to be humpback.

Tell the mothers of the families you are called upon to attend that a swelled joint may be the commencement of a serious disease; and thus aid in the diffusion of knowledge which, if properly interpreted, will save more children from deformity and lameness than all the apparatus ever devised for their relief.

The synovial membrane contains absorbents; it has a limited neural distribution, and its vascular supply is good. After the lesion of chronic synovitis has existed for some time, however, its absorbent power is greatly interfered with. In simple hydrarthrosis, where there are unimportant changes in the synovial secretion, the absorbents still act, and one of our greatest hopes in the treatment of chronic synovitis is that the free surface of the membrane is not disintegrated by the fungous masses. Upon this basis, in certain cases, we make use of compression. For this purpose we use a silk elastic anklet, compressed sponge or a firmly-applied flannel bandage, which latter should be frequently readjusted. We also use such remedies externally as may excite the absorbents, if there be any left, to greater action. Oleate of mercury, iodine, etc., are useful; so are blisters and friction by the hands. A gentle, but persistent surface massage seems to possess the power of assisting in exciting the absorbents. If the joint shows a higher temperature than normal, avoid the use of excitants and apply ice or other cooling applications, continued, if necessary, for a long time. Other remedies, such as the actual cautery, etc., suggest themselves; but they will occur to you as you study your cases, and read, if necessary, the authors whose contributions are so excellent. Study your entire armamentarium of internal remedies, and do not confine yourself for a long time to any one remedy. Iron, the hypophosphites, the various iodides, mercury, etc., are indicated, depending on the actual condition, the hereditary influences and tendencies. I have known mercury and iodide of potassium to accomplish excellent and rapid cures in cases with a syphilitic taint. I have known it to fail in many other cases. Iron is almost always indicated and a favorite prescription of mine is the plain tinct. ferri chlor., in large doses, combined with an equal quantity of glycerine. It seems in many cases to aid the plastic power, more than any other preparation I have used. But I might add a long list of preparations with which you are familiar. I will again state, before commencing the subject of mechanical treatment, that each case is a study in itself, and that you must determine, by experiment if necessary, the remedy or remedies necessary to aid you in the all-important element of mechanical support.

Whatever value traumatism may have as the *cause* of chronic joint disease, such as we have been speaking of to-day, there can scarcely be anyone who doubts its relation to the progress of the lesion, when once it is established. It is, at least, my firm opinion, that when a joint or any of its essential structures become diseased, we should adopt means which will, without doubt, remove the element of constant traumatism, produced by locomotion, etc., and which will protect the vulnerable tissues under all circumstances. This support, at the same time, should not materially interfere with exercise and locomotion. How shall we apply this general principle to the conditions we have been studying to-day? Nature rarely presents a pathological condition without, at the same time suggesting, if not a remedy, indications for its use. We learn from the pathological state and a study of the clinical features, how to apply the means of relief. Let me recall what we have said about *chronic synovitis* and see what it suggests.

In chronic synovitis there is almost normal use of the joint, with the fatigue after exercise. We should not, therefore, immobilize, for nature does not make the attempt. We should meet the indication, fatigue after exercise, and apply an apparatus which gives absolute joint protection.[1] In other words, when the patient is sitting there need be nothing but protection against accidental traumatism; but when the patient walks this protection must be absolute, and the means by which it is to be obtained *should act promptly and without the exercise of volition on the part of the patient.* These, in brief, are the indications for the mechanical treatment of chronic synovitis of the ankle-joint.

Bear in mind, for it is very important, that you should always arrange your mechanical aids in treatment of chronic joint disease so that it shall *always act independently of the patient's will.* Make your protecting instrument, practically, part of the patient and part of the affected joint and limb; adjust it so that the patient can not move or step without making the support available; apply it so that whether sitting or standing, running or walking, whether the danger comes from the patient's own indiscretion or from some accidental cause, the joint —the vulnerable point—is always protected. I have seen so many inefficient methods adopted for these cases, so many truly useless procedures, that I feel that I cannot speak too strongly upon this point. It is true that anything which gives partial support may produce temporary relief; but, when the patient feels this relief, he at once gives the affected joint more liberty, and, being not fully, or so to speak, not automatically protected, a speedy relapse ensues. Do not *seem* to do your work in orthopædic surgery, but always be sure that you accomplish all that is required when you use a support. Devise or prescribe the instrument yourself, and see that it is so constructed and applied that the principle involved is carried out. Of course, you must rely somewhat upon the discretion of the patient or his nurse; but you can, by study and effort, so construct and so apply your supports that their use may be reduced to the adherence to a few simple rules, and you can soon tell whether or not these rules are obeyed. If they are not I would candidly advise you to dismiss the patient, and devote your time to some other and more profitable employment.

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[1] See "The Mechanical Treatment of Synovitis of the Knee-Joint." By C. Fayette Taylor, M.D. *N. Y. Medical Journal,* July, 1873.

Mechanical aids are essential to the scientific treatment of chronic joint disease. They will prevent deformity in many cases if they can do nothing more. In others, they will, where anchylosis is inevitable, permit you to elect the ultimate position of the joint. And you should always have at hand the facilities for adapting the instrument to the indications, and for making the instrument actually do the work required of it. You should master thoroughly the detail; you should always know more than the instrument maker, not necessarily about the manufacture of the apparatus, but about all other points. You should no more go to an instrument maker for advice than you would to a druggist for instruction. You should not send a patient to an instrument maker for treatment, any more than you would recommend a patient for treatment to a pharmacist. You should prescribe your instrument upon a rational basis, just as you would prescribe for an acute disease; and you should be able to tell the instrument maker, if the instrument is faulty, where the fault lies, or, better still, you should be able to correct the fault yourself. Upon this basis I know of no more satisfactory or pleasant work than orthopædic surgery. But, under any other conditions, it is the most unsatisfactory kind of work. The profession must sooner or later appreciate this and when they do orthopædic surgery will be properly understood and appreciated and that kind of talent will be attracted to it which will make its future brilliant. For there is more to be learned, I feel, in orthopædic surgery than in any other branch of surgery; and hence, there is more to be gained by those who diligently follow it, and intelligently study its problems.

To revert more particularly to chronic synovitis again, I said, and I again repeat it do not immobilize the joint in *chronic synovitis.* If you do, the joint soon becomes stiff and useless, and the closely observing friends of the patient will inform you—and they will state a fact—that the joint is in a worse condition than before you applied the apparatus. Use of the joint in chronic synovial inflammation, therefore, is indicated. Motion, without pressure, is plainly demanded; in other words, we must *avoid traumatic contact of the vulnerable surfaces.* To accomplish this we must produce a certain amount of traction, but we must not prevent free movement.

There are no muscular contractions to overcome; there is no deformity of the joint to remove; there is no pain caused by interarticular pressure in chronic synovitis. The idea should be to produce just enough traction upon the joint to transfer the weight of the body to some other point, giving the joint full liberty when the pressure caused by the weight of the body is not present. Clinical experience teaches, in ankle-joint synovitis, that if we apply an apparatus that just prevents the foot from touching the floor when the patient walks, we place the joint in the best possible local condition for repair.

I may say here that I have been disappointed with all instruments that I have seen, that have been devised for the treatment of ankle-joint disease, whether synovial or osseous, that act upon the ankle-joint alone. In order to perfectly protect the ankle-joint we must limit more or less the movements of the knee-joint. If we wish to immobilize the ankle-joint in locomotion we must also control the knee. This fact we can, in some cases of synovial disease, ignore; but in the vast majority of cases we cannot ignore it; and hence, we must include the knee-joint in our support. But where shall we obtain our counter support? If we are going to actually protect the ankle we must not depend on adhesive plaster and its attachment to the skin for our counter support. This is one of the fallacies that I have tried to combat. Adhesive plaster forms a very excellent and, indeed, the best means of applying direct traction to a distal part of the body, where we have a fixed point to pull against, as in hip disease or knee disease, or even in ankle disease, if we apply our adhesive plaster to the parts beyond the ankle-joint. We cannot make a reliable fixed point with adhesive plaster. We must go to the perineum for our fixed point, to the tubera ischii, just as we do in hip disease; and we must make our support so that we can control the knee-joint if necessary, and actually support, protect or apply traction to the ankle-joint as indicated. In chronic synovitis of the ankle-joint, and in chronic osteitis, also in some other cases we can allow the patient to bend the knee when he sits down. As I will show you, there is a peculiar arrangement at the hip and knee in the apparatus I use, by which the patient can flex the knee-joint and the hip-joint as he sits down, and this mechanism locks automatically in the straight position when the patient stands erect or walks.

Here, gentlemen, is the instrument I use in ankle-joint disease; it looks formidable when removed from the patient, but when adjusted properly it is one of the most comfortable and efficient I have ever used. It is composed of six essential parts: (1) The pelvic band *A B*, which enables us to make the fixed perineal point of support *E E*. (2) The automatic hip-joint movement *C* which admits of flexion when the patient sits down, and which locks without aid when he stands. (3) The thigh part *G* in which we place the extension rod. To this thigh-piece we attach a plate *H* for loose lateral attachment to the thigh. (4) The knee-joint, with its automatic "snap" *J*, which always locks when the patient stands up and commences to walk. (5) The calf part *N* which, like the thigh-piece, has a band and leather lacing to afford lateral attachment. (6) The

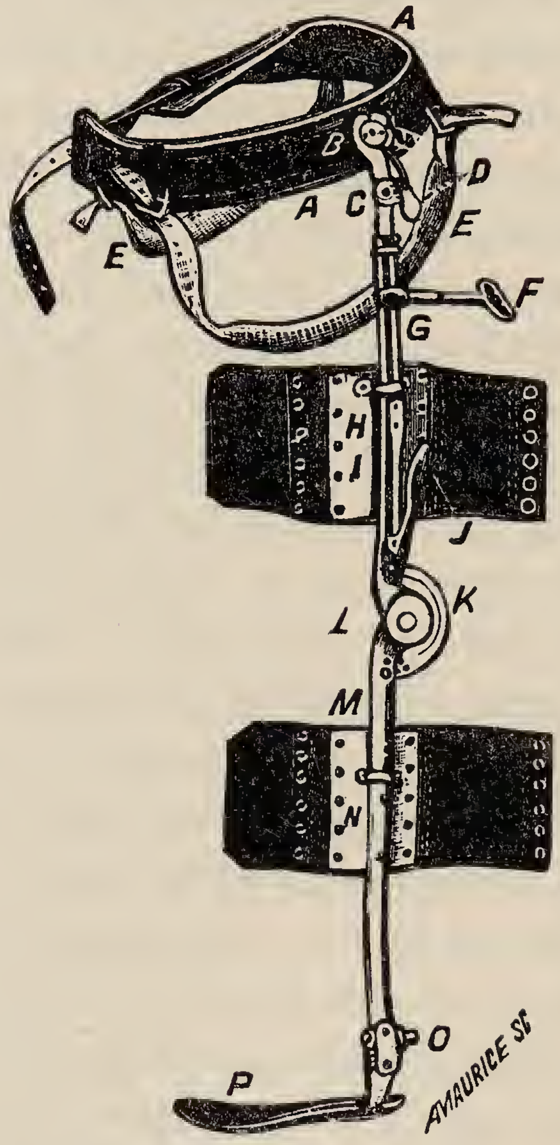

FIG. 1. SPLINT FOR THE TREATMENT OF ANKLE-JOINT DISEASE

ankle-joint *O*, with a worm and screw, which enables us to place the foot-piece *P* in any desirable antero-posterior position (see Fig. 1).

This instrument is modified from one introduced by Dr. Taylor, for chronic synovitis of the knee-joint. Dr. Taylor's instrument is pictured in his article on "The Mechanical Treatment of Synovitis of the Knee-Joint," already referred to. To it I have added the extension rod *C*, and the pelvic band *B*, the automatic joint at the hip *C*, and the worm and screw, controlled by a key at the ankle *O*. The joints at the hip and knee permit the patient to sit in an easy position, and when the patient stands they each lock automatically, and form a continuous splint. Protection, with traction, if desired, is thus always secured when the patient sits, and traction is always present when the patient walks or stands.

The instrument is well adapted, in a mechanical sense, to the treatment of either chronic synovitis or chronic osteitis of the ankle-joint. The modifications necessary to meet the indications in these two conditions are made at the ankle-joint. For chronic synovitis we find just how much motion the patient should have, and then, by a "catch" in the sidepiece of the foot-plate (this is not shown in the plates) we limit the flexion and extension of the foot. We then apply the instrument, and generally without any adhesive plaster whatever. The instrument fits the contour of the limb, and we adjust it so that the foot-plate comes closely in contact with the sole of the foot, before the shoe is applied and with the pelvic band just below the anterior superior spine of the ilium. Then lace the calf piece *first;* then apply the shoe *over the foot piece.* You will be surprised, if you make the instrument fit, how easily the ordinary shoe goes over this foot-piece. Then lace the shoe quite snugly; then lace the thigh-piece;

finally buckle the pelvic band, and secure the perineal pads. Now make gentle traction, with the key just enough to carry the foot-piece, enclosed by the shoe, slightly away from the sole of the foot. Place the patient on his feet. Almost the entire weight of the body falls on the tubera ischii when the affected ankle-joint is used in locomotion. The knee-joint is stiff, or not as you would wish, for, by removing a couple of screws, the automatic spring can be taken off. The ankle is protected against all ordinary traumatic influences, and the apparatus is almost as much a part of the patient as though he had

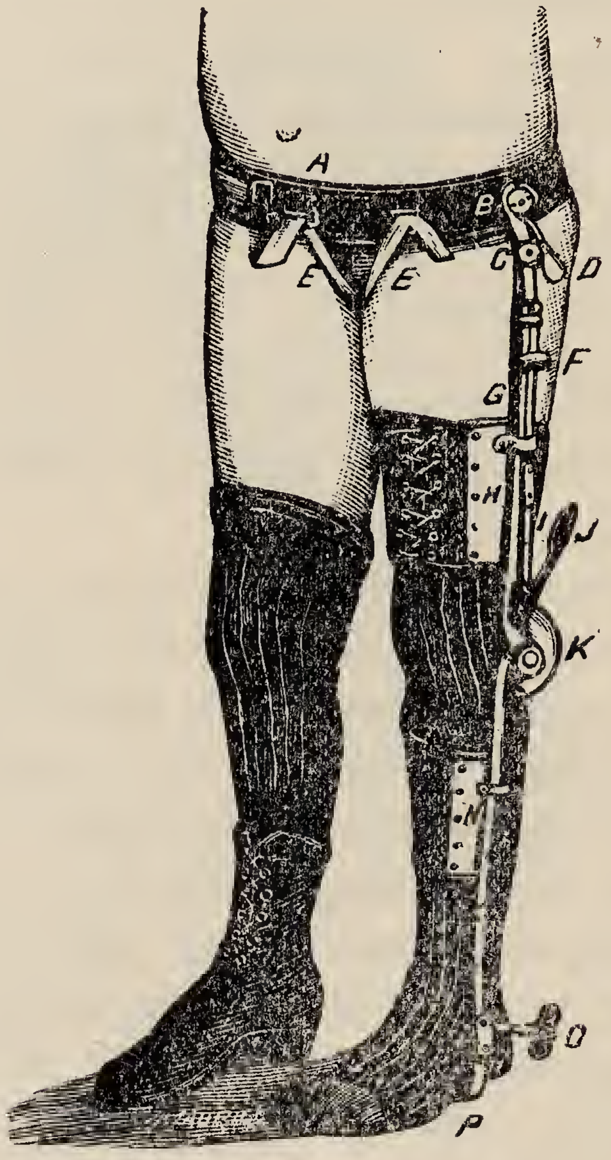

FIG. 2. SPLINT APPLIED IN THE TREATMENT OF ANKLE-JOINT DISEASE

been born with it. Whatever the patient does, the support is constantly acting, and no voluntary effort is needed to protect the inflamed joint surfaces. The risk of traumatism is reduced to a minimum, and you can now satisfactorily commence your systemic treatment. You can always control the amount of motion at the ankle-joint; and at night, if you wish to protect your patient against accidental traumatism keep the instrument on and apply a bandage in place of the shoe, or adjust some simple form of ankle-joint splint. You ought to insist that the patient should never walk without the support; for if you permit any discretion in the matter, you might as well have no rules at all. If you say that the patient can walk without the support occasionally in the treatment of chronic synovitis, he will sooner or later make it his habit to reverse your rule, and wear the support occasionally. In chronic osteitis, however, there is very little necessity for this rule. The comfort afforded by the apparatus so far exceeds the discomfort occasioned by its use that the patient will much prefer to wear the support.

In using this instrument for chronic osteitis of the ankle you not only have a different pathological state from that found in chronic synovitis, but you must also make the modification necessary to meet it. You have no malposition of the joint in chronic synovitis, and hence, you can use the apparatus with more or less antero-posterior movement at the ankle. But in chronic osteitis we find reflex muscular spasm, malposition, either antero-posterior or lateral, pain and much greater difficulty in locomotion. Still, exercise in the open air is indicated, and joint protection is much more urgently demanded. How shall we make this instrument meet the indications?

Let me say here, for your future guidance, in the treatment of chronic articular osteitis, that you should always adapt your apparatus *exactly* to the deformity, whatever it may be, and that you must, by gradual and very gentle changes, restore the joint to the normal position, or that position, whatever it may be, which you seek. For ankle-joint osteitis I have introduced an antero-posterior worm and screw (*O*, Figs. 1 and 2). We can then, by a key, turn the foot-plate into a position of equinus or calcaneus, and so adapt it to the deformity; for there is no lateral malposition of the ankle-joint itself, and we can bend the side-bar to meet the secondary tarsal malpositions. After we have adapted the foot-piece and the side-bar to the actual deformity, we go through the same process as that just now described—viz.: We lace the calf-piece first, then adjust the shoe, then lace the thigh-piece, and lastly, arrange the pelvic band and perineal pads. We can reenforce this laced shoe with adhesive plaster, if necessary, however, and then make very thorough traction, applying considerable force, and in this way producing that peculiar avoidance of injurious contact which is so grateful to sufferers from chronic osteitis of the joints.

Patients who could not walk before the support is applied will generally walk well after this apparatus is properly adjusted, without any further aid. They do not need crutches or cane. It is only necessary to make the sound limb artificially longer with a high-soled shoe, and advise care in locomotion at first. You do not have to urge these patients to walk; they soon become very active —as active, indeed, as children without joint disease.

Gradually you will change the position of the foot-plate, and the foot will follow it. In a few days you will see the ankle-joint in a good position, and the progress toward recovery fairly commenced. The instrument, as adjusted, but without the shoe or traction, is shown in Fig. 2.

Before giving you some practical demonstrations, I will briefly refer to some other matters: 1st, regarding abscesses: their presence does not interfere with the use of this support. Bad cases, with œdematous infiltration, sometimes improve very rapidly under its use, and you can very easily arrange the support so that it will not produce any discomfort. 2d. If pain be very severe it is advisable to apply adhesive plaster, and to keep the patient in bed, where the proper local treatment can be carried out. 3d. Give a few *positive* rules for the guidance of your patient, and insist upon their being obeyed.

The question of operative interference, excision, etc., is applicable to other joints as well as to the ankle, and I will consider these and other matters upon some subsequent occasion. I may say, however, that I have seen very few cases of ankle-joint disease where I deemed excision or amputation justifiable in a child.

ON THE PRINCIPLES OF THE MECHANICAL TREATMENT OF HIP-JOINT DISEASE[1]

Read before the American Orthopædic Association at its third annual meeting, in Boston, September, 1889.

HIP-JOINT disease, especially in childhood, may be described as a chronic tubercular lesion of the coxo-femoral articulation, with the major signs and symptoms in the initial stage suggesting a primary osteitis rather than a synovitis. The clinical history and the course of the disease, and the pathological conditions found both after excision of the head of the femur and after death, all tend to emphasize this statement. In discussing the principles which should govern us in the treatment of this lesion, I shall accept this as my text, for without some generally understood pathological basis it would be difficult to formulate any definite principles of treatment, either mechanical or otherwise.

Nor can we proceed intelligently without a glance, at least, at some of the more important of the signs and symptoms of the disease. And we will briefly consider some of them.

As is well known, an insidious and generally unexplained limp is almost always the first sign that claims our attention. And this very suggestive evidence of the change that is going on in the articulation is always, I think, accompanied by a still more suggestive and important indication—viz., an instinctive and involuntary muscular protection of the articulation. Long before pain or any apparent deformity appears these two important indices of hip-joint disease are present; and it is now, I think, generally accepted that the condition which accompanies these two signs, when typically expressed, is that of the first apparent stage of hip disease. My own experience has proved, and every year the proof has grown stronger and stronger, that this limp and this instinctive and involuntary muscular joint protection are necessary to establish a diagnosis of hip disease in the earliest stage. We all recognize that many other conditions may produce a limp and the muscular signs which simulate hip disease, but the differential diagnosis is not on the whole difficult, owing to the peculiar and expressive character of the involuntary muscular protection of the joint.

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[1] *The New York Medical Journal*, for November 23, 1889.

It is not assuming too much, I am sure, to say that these two signs—the limp and the instinctive muscular protection of the joint—have a peculiar significance. The limp may be interpreted as an instinctive effort on the part of the patient to avoid the increased inter-articular pressure produced by the simplest act of locomotion; and the second, the muscular protection, indicates that the muscles acting upon the affected articulation, guard the joint when the joint lesion is exposed to even the slight traumatism of passive motion.

The whole expression of the articulation in the first apparent stage of hip disease is, if I may be allowed the expression, one of joint apprehension under certain tests and in certain conditions. It is very like the apprehension which accompanies the more advanced and painful stage of the disease, when any and every movement of the joint produces an agonizing pain—only the acute pain is absent, and the apprehension is less marked. In short, it is the first stage of a chronic disease, unmarked by pain or fever, and the whole expression of the affected joint under examination is that of a protest against traumatism, both the limp and the acute but slight muscular resistance crying out against the simplest functions of the normal joint—viz., gentle locomotion and a natural degree of articular mobility.

It is well known that this first stage of hip-joint disease —the stage of vulnerability—where the trouble has not fully developed, but where Nature has kindly given us ample evidence of the extensive mischief that is impending, the limp referred to as occurring in this first stage may disappear. The rest and protection which the patient instinctively gives the affected joint are sufficient in some cases to remove the limp and to modify the involuntary muscular protection. But the joint still remains vulnerable, and a major traumatism, like a fall or a blow, or even the constant repetition of the minor traumatism caused by locomotion, re-excites the latent pathological condition, and the limp returns and muscular protection again becomes pronounced. I have known this to occur seven times in one patient before the symptoms became so pronounced that a diagnosis was made, the surgeon in attendance being unfamiliar with the fact that a remission of the limp was likely to occur in the early stage. It has happened several times in my experience that the limp has disappeared without active treatment while the patient was under observation. I have watched these cases with much interest, and I have found that *the muscular protection of the joint did not wholly disappear, as the limp did.* It may be worth while to say that temporizing in these cases proved a mistake, for in every case, after a lapse of weeks or months, and in one case of two years, the symptoms reappeared with redoubled energy, and the lost time was not always wholly regained. In short, so long as there is a true pathological and involuntary muscular protection of a joint in the first stage of tubercular hip-joint disease (and also, I feel sure, in the second and third stages as well), we must imitate Nature's method and place the joint under conditions where it can receive no major or even minor traumatism.

And this, it seems to me, must be the fundamental principle of mechanical treatment—viz., to protect the affected joint—and to secure for it that which Nature from the very beginning of the lesion attempts to secure—relief from traumatism. In the first apparent stage, that of the initial vulnerability of the joint, where the limp is slight and the muscular protection is only, perhaps, just perceptible, adequate mechanical protection of the affected articulation is just as imperative, in my experience, as it is when the disease is well defined and fully developed. All of the cases that I have watched where rest—simple rest in bed—has been pursued as the only treatment, have failed to obtain permanent relief. I have watched cases of this kind long enough to know that those which I have thought were cured, and if I had been writing an essay at the time they passed from active observation I should have reported as cured, have all, after a longer or shorter time, relapsed, if prompt relief from traumatism was not given by apparatus efficiently applied.

From a mechanical standpoint, therefore, the first object of treatment should be to modify or to absolutely remove, if possible, traumatic contact of the opposing joint surfaces in hip-joint disease; for, however much the medical profession may differ regarding the influence of traumatism as applied to the ætiology of this disease, I think none will deny its baneful influence after the lesion is once established in the joint. The second object, having placed the patient under the best possible local (joint) conditions for repair, is to surround the patient by those hygienic influences and to administer those remedies which will aid Nature in eliminating a tubercular disease. On this point I need not dwell. I think this association as a unit will agree that a patient with tubercular disease in any part of the body will do better, for example, in the pure air of the Adirondacks than in the wards of a hospital. And if the patient has hip-joint disease he will do better walking about in the fresh air in an efficient protective apparatus properly applied than if he be confined to bed with or without a protective splint.

To return to the first object—that of securing a modification of the traumatic contact between the surfaces of a tubercular hip-joint. We have seen how Nature instinctively protects the diseased joint in the first stage. In dealing with the more difficult problems of the second stage (that of deformity) it will be well if we again look to Nature's methods and see what she attempts to do; and we will be wise if we imitate, if we can not always accurately follow, her example.

The first apparent stage of hip disease has been referred to. This early initial stage is one where, in my experience, it is comparatively easy to secure a perfect cure of the disease without deformity or without any serious modification of joint mobility. If the first stage, however, be permitted to go unchecked, as it usually does in the absence of any urgent symptom, the second stage, that of deformity, appears sooner or later. The signs rather than the symptoms of the stage of vulnerability have passed unheeded, and Nature has narrowed day after day, week after week, perhaps even month after month or year after year, the arc of femoral movement in all directions, until in many cases the results are apparent ankylosis of the articulation with a marked deformity. This deformity is apparent first as a flexion; then, as a rule, follows abduction, and this is usually accompanied by a rotation outward of the femur. After a time adduction and rotation in the opposite direction may ensue, but flexion is always present.

It is not only possible, and in many cases more than probable, that a good result may be obtained with free joint motion in the second or deformity stage. But the golden opportunity has passed when the limping gait becomes emphasized by a deformity, or, in other words, when the muscles, having placed the joint in the position of the least traumatism and of the greatest ease, instinctively and involuntarily protect, to the best of their ability, the diseased part or parts.

In the early part of the disease, and oftentimes in the second stage (before true adhesions have formed), if we administer an anæsthetic and pursue its effects until a profound narcotism is produced, we find there is little or no resistance to full and free joint motion. As soon as we temporarily interrupt the reflex arc, and relieve the muscles of their duty, we find the muscular evidence of hip-joint disease absent—*i.e.*, there is no resistance to free joint movement and there is no deformity.

Nature, in short, in hip-joint disease is producing in her own way an involuntary muscular splinting of the joint—the tense and shortened muscles, in a typical case, making an almost perfect coaptation splint. But there is this difference between the conventional coaptation splint of surgery and the involuntary muscular splint of hip disease: The force of the former is exerted, as in the treatment of a fractured thigh bone, for example, *laterally*, or in a direction which lessens the circumference of the splint. On the other hand, in hip disease the force of the muscular splint is exerted *longitudinally—i.e.*, in the direction of the long diameter of the contracted muscles, or in that direction which throws the head of the femur against its socket. This muscular splint in hip-joint disease simply avoids the major joint injury of motion and pressure combined by substituting for it another form of articular injury—viz., an interarticular pressure which limits motion and retains the joint by means of this interarticular pressure in that position which, unaided by art, inflicts the least injury to the diseased joint. The limitation of motion in the joint is accomplished only at the expense of an intensified joint pressure—a pressure which is much further increased by any form of mechanism which uses the femur as the long arm of a lever to correct the deformity.

When the second stage is reached, locomotion becomes very difficult, the whole body swaying around a deformed joint, and each successive traumatism—even that of walking with the aid of crutches, and surely that of an unscientifically applied apparatus—produces a fresh grazing field for the tubercular bacilli. The transition from the second to the third stage (that of joint disintegration) is so gradual that it is difficult to draw the line. But, sooner or later, if the disease be not arrested, a more or less rapid joint disintegration ensues. The femoral head becomes smaller and smaller, the acetabulum becomes concentrically enlarged, perhaps perforated, abscesses may form, and finally, after the disease has run its course, the ultimate deformity and disability with which we are all familiar appear. Even in this last stage the muscles are tenacious of articular control and refuse to grant the full movement the altered joint surfaces permit. The muscles are only quiescent when in those exceptional cases, and after years of struggle, actual ankylosis occurs, or the tubercular process is wholly ended.

It seems perfectly apparent to me that the deformity of the second stage is due to an involuntary muscular action, and that the deformity of joint disease, irrespective of the question of whether there is a synovial effusion or pus within the joint capsule, represents Nature's position of greatest ease. This muscular coaptation splint is Nature's only way of modifying traumatic contact of the opposing joint surfaces, and this modification is secured by a change of position. It would seem that this muscular effort or splinting is more apparent in some cases than in others.

At the clinic of the New York Orthopædic Dispensary and Hospital we see quite often cases of very considerable and sometimes very tense accumulation of pus and tubercular material within the capsule of the knee and ankle joints where there is little or no interference with articular mobility. The deformity in these cases exists only in joint outline. The mechanical relations of the opposing joint surfaces are not interfered with and there is little or no muscular effort, either voluntary or involuntary, to control or limit the movements of the affected articulation, and pain is wholly absent. The signs of disease (there are no symptoms present at all) which present in this class of cases at the knee and ankle do not seem to indicate an osteitis; on the contrary, the synovial membrane is distinctly diseased and the lesion is probably confined to this membrane, and perhaps superficially to the cartilage also.

In this class of cases, where the synovial membrane is apparently much affected and the osseous tissues have escaped major injury, Nature makes no effort to limit joint motion by muscular action. I have attempted elsewhere to explain why this is so.[1] I merely desire on this occasion to call attention to these two classes of chronic tubercular joint disease—the synovial and osteitic —and to say that where the synovial membrane is primarily and solely affected at the knee and ankle, the mechanical treatment should be different from that which will be mentioned in this paper on tubercular disease of the hip.[1] And I also wish to call attention to the fact that in hip-joint disease we are always met by the initial symptoms of a bone lesion rather than those of a simple synovial disease. Whether we have chronic synovial disease at the hip in the early stage of the trouble, it is impossible, from a clinical standpoint, to say. In the well-marked cases of uncomplicated synovial disease at the knee and ankle, the only evidence of the lesion, as already remarked, is an objective one—a synovial swelling —a sign that for anatomical reasons is not always available at the hip.

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[1] "The Ætiology and Pathology of Chronic Joint Disease." *A Series of American Clinical Lectures*, Vol. iii, No. 6.

The cases of synovial disease at the knee and ankle are, however, much less frequent than those which at the same articulations from a muscular standpoint are the perfect analogue of the muscular and deformity conditions found in hip disease. In these latter cases there is no essential change in joint outline, no evidence of a distended or swollen capsule or synovial change, but rather a spurious ankylosis induced by the same muscular conditions which produce the second stage of hip-joint disease. Here, in this latter or osteitic class of cases at the knee and ankle, it is easy to interpret Nature's effort to modify the traumatic contact of the opposing joint surfaces, and to understand that in the osteitic condition Nature abhors motion and uses every effort to restrain it, and to restrict it within certain limits. Within the limit set by Nature, motion I believe to be harmless, and hence it is that it seems wrong to immobilize a knee joint, for instance, with simple, uncomplicated synovial disease of the tubercular variety. But if we attempt, in the osteitic form of the disease, to pass beyond the limit set by Nature, or if we try by unscientific means to secure a better position for the distal half of the joint when Nature by her every expression says "No," we ignore the valuable lesson which she teaches us and inflict more injury, in many cases, upon the diseased articulation than all our boasted orthopædic science can relieve. And a very wide difference of opinion among both orthopædic and general surgeons would be bridged, I think, if the fact were recognized that where there is some or even a little motion at a diseased joint, true immobilization is not necessary. But motion under these circumstances should be controlled within the limit set by Nature. A joint that is practically immobilized by the muscular coaptation splint will recover motion under the absolute fixation of traction, as has been proved very many times.

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[1] "A Lecture on the Prognosis and Treatment of Ankle-joint Disease." *Annals of Anatomy and Surgery*, May, 1882.

I have seen on many occasions, especially in the large general hospitals of this and other countries, irreparable injury inflicted upon a slightly diseased hip joint by useless manipulations and tests, both with and without anæsthesia. I have known apparently curable cases to be rendered hopelessly incurable by an ill-advised attempt to rectify the deformity of hip disease in the second stage, when there was little or no thought bestowed upon the plain expressions, muscular and otherwise, of the pathological condition. The absence of pain is too often made the criterion of whether a patient with hip disease should be subjected to certain tests and certain forcible methods of treatment, the decision generally being that if there is no oral expression of pain the joint would stand almost any desired force without injury. My own experience leads me to say that very frequently the patient in whom there is an entire absence of the oral expression of pain is the one who should be the most carefully and conservatively treated, and about whom should be thrown the most careful safeguards against traumatism. No one, for example, would be likely to advise the use of forcible measures to remove deformity in a case of hip disease when the patient was in the exquisitely sensitive stage; and yet many patients, where this acutely sensitive stage is but a step removed, are subjected to unnecessary and in many cases to unscientific measures of treatment. My experience leads me to say that if there is a condition where preventive measures can be applied, where much mischief can be prevented, where it is our duty to anticipate the next bad phase of the disease and to avoid it, it is in hip-joint disease and allied affections; and he who passes by unheeded the plain indications, or he who with a limited experience maintains that operative surgery can accomplish in the treatment of chronic joint disease more than Nature, conservatively assisted and protected, has yet to learn the pleasure that can be derived from a patient and diligent conservative treatment of a diseased joint.

If, then, we are to aid and imitate Nature, rather than to force her, in the treatment of hip-joint disease, and we have interpreted aright the various phases of this condition, there need be no hesitation as to the course to be pursued.

In the first stage—that of the earliest noticeable vulnerability of the disease—when there is a slight limp and an almost normal arc of motion, it is comparatively easy to protect the articulation from traumatism, because nature affords a wide range of movement within which, with scientific mechanical protection, it would be difficult to apply any traumatism to the joint. There is no articular deformity present and the muscles are not tense and irritable, except as the vulnerable parts are exposed to contact or pressure. While it would seem that the mechanical treatment of these cases is simple, it is best to take no risk, but to make the artificial joint protection as complete as possible. The major difference in the detail of treatment between the first and second stage is that while in the second stage we must overcome or greatly modify the deformity before permitting locomotion—and a shorter or longer period of recumbency is necessary to accomplish this—in the first stage we have no deformity and the patient can at once assume the upright position and walk about in a properly constructed apparatus. When the deformity of the second stage is removed, locomotion—also with the aid of proper apparatus—is to be advised, and within certain limits encouraged.

It is therefore the second stage of hip-joint disease that demands our principal attention, for in this stage we are met by the most expressive evidence of the disease and the most important problems of treatment, and it is in this stage that a single error may undo the work of previous weeks or months.

Let us, for example, take a case of hip-joint disease in the second stage, where there is a marked deformity and where there is a very considerable modification of articular mobility, and attempt to determine the indications for the mechanical protection of the joint. We need not stop to consider whether pain or abscess are present. The indications from a mechanical standpoint are the same whether these conditions are present or absent.

The principal apparent expression of the joint lesion is the deformity. It is so important, both to the mind of the patient and his friends and, in many instances, to the surgeon himself, that the grave lesion of which it is the simple index is lost sight of, and the mistake is very frequently made of hastening the reduction of the deformity at the expense of the articular disease itself. It is here that errors are most frequently made, and it is here that we should be prepared to use, with all patience and diligence, the most scientific and efficient means at our disposal. I have often remarked that if hip-joint disease was unaccompanied by its expressive deformity it would lose its principal terrors for both the surgeon and patient; and he who would strive for the best attainable result in this formidable complaint must always remember that hip-joint disease is no trifling affair, and that it were better—far better—to use no mechanical treatment at all than to attack the disease by an immature and unscientific effort, by apparatus or otherwise, simply to modify one of its signs. I have no doubt at all that much irreparable mischief and that many avoidable abscesses and death itself have been occasioned by meddlesome mechanics in the treatment of hip-joint disease, and I know of no problem in orthopædic surgery, unless it be that involved in the mechanical treatment of true lateral curvature of the spine, which requires the expenditure of more thought and effort than that which claims our attention in the treatment of hip-joint disease in the second stage.

Whatever may be the primary cause of the deformity in the second stage, no one, I think, doubts the important part assumed by the muscles in the production of the malposition, as well as in maintaining it after it has once been produced. And no one who has examined many cases of hip-joint disease in the deformity stage can doubt that the principal resistance to the reduction of the deformity lies in the muscles, which are on guard all the time to protect the joint from undue movement, which, in joint language, is synonymous with the slightest traumatism. The first and the most important step in the treatment of the disease is to remove this element of trauma from the joint. This simple statement underlies the whole problem of the value of the necessity of mechanical treatment, and I have no hesitation in predicting that that method which best subserves this indication is the one which will outlive all the others.

If in the second stage the thigh is flexed and abducted and we attempt by manual effort to overcome either deformity, even if guided by the intelligent hand of the most experienced orthopædic surgeon, we find that the muscular resistance can not be overcome by any ordinary effort, and that nothing but an extreme degree of force, which inflicts intense pain, will modify the acquired position of the femur and bring it into the so-called straight position. There can be no disputing that this resistance to the reduction of the deformity, and the pain which we inflict while doing so forcibly, mean that the joint, in its sensitive and apprehensive condition, demands adequate protection, and they mean as well, also, that he who would afford his patient the most complete relief and the best attainable result will study these and other things which the joint almost talks to us about, and make available the lessons which they convey.

It seems perfectly apparent to me that a joint in the condition above described needs, as a first step toward recovery, an essential modification of the traumatic contact between the opposing articular surfaces[1]—not in the position we wish it to be in, but in the position which Nature has selected.

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[1] Dr. E. G. Brackett, of Boston, has proved that in childhood distraction of the hip joint is an evident fact. Vide *Boston Medical and Surgical Journal*, Vol. cxxi, No. 17, October 24, 1889.

No one who has seen a child suffering with the agonizing pains of hip-joint disease, who has been crying night after night with the lancinating neuralgia of an inflamed hip joint, go to sleep soon after the thigh has been held quietly but firmly in the chosen position of the disease, can doubt the value of the relief afforded by manual support and protection intelligently applied to these cases. The certainty of the relief afforded by this simple test is in no way diminished by suddenly or even gently relaxing the support given by the hands of the operator. The quick and apprehensive cry that follows, and the shiver that runs through the frame of the little sufferer, give positive evidence of the value of properly applied protection in these conditions. It is, I think, our uniform experience that when pain is present in these cases it affords an infallible guide and an unerring indication as to treatment. My own experience goes still further, and I am prepared to say that if pain were present uniformly in the second (or even the first) stage of hip-joint disease, our results would be better and sooner attained. My experience further demonstrates that those who aim for the best attainable results are those who treat their patients as if there were a slight pain present all the time, and who follow them up with the same careful attention they would bestow upon a joint that is slightly sensitive; for the less sensitive joints are none the less removed from the dangers that threaten the painful ones, and the feeling of false security that comes to both the patient and the surgeon, when pain is not present, leads to many errors of treatment and to many bad results. In a well-applied portative apparatus, embodying correct principles, the patient is apt to forget that he has any joint disease, and the surgeon is prone to hope, if not to think, after a while, that the worst is over and that a favorable end of the disease is very near. It is under these circumstances when the symptoms are latent only, and when the disease, though either stationary or progressing toward recovery, is far from the desired end, that it is the duty of the surgeon to in no way relax his attention, for the period of convalescence in hip-joint disease is in many respects the most perilous.

I think I am wholly warranted in saying that, whether pain is present or absent, we should adopt from a mechanical standpoint the best-known means of modifying, as a first and as the most important of all considerations, the traumatic contact of the affected joint surfaces.

In the experiment referred to above, when manual support is given to a painful joint, it is found that simply supporting or fixing it by the hand or hands gives little or no relief. If the lever principle is added and it is attempted to fix the joint in that way, the pain is increased. But as soon as a traction force is applied—the body forming a counter-traction—relief is almost instantaneous. And it is found, in typical cases, that the pain returns just as soon as the traction is relaxed or modified. This experiment has been made by us all, in many cases and under all kinds of circumstances, and it is among the well-demonstrated facts of orthopædic surgery. If, now, continuing our tests we attempt to alter the relation of the joint surfaces to each other—*i.e.*, attempt to correct the deformity even while exerting this traction or pulling force—we find that the pelvis moves with the femur, so strong, so active, and so incessant is the muscular splinting of the joint. We also find that when the change of position involving the use of a lever is attempted the pain at once returns, and the more force used in making traction, save in the position which gives the patient comfort, the more the patient complains and the greater is the pain inflicted.

Let us pursue our experiments with manual traction still further. Let us keep up the traction for an hour or two, or even longer, in the direction which brings relief, as I have done on many occasions when, with no apparatus attainable, sleep could only be purchased by manual traction. What happens? Little by little, as the trauma, which has been exerted upon the joint surfaces for the preceding weeks or months, is relieved, the muscles relax their tension and it becomes possible to obtain an increase in the joint motion without inflicting pain. After traction has been kept up persistently and intelligently for an hour or two under these circumstances, the patient will even sleep for a short time without any traction at all. But if we desist entirely and allow the limb to again resume its chosen position, the muscular tension soon returns and the joint will become as rigid and as painful as before under the influence of the muscular splinting which is so characteristic of both the painful and non-painful conditions.

These experiments have doubtless been unconsciously made by us all, not only with the hand, but by properly constructed and applied traction apparatus. It is an almost daily experience for us all to reproduce with traction apparatus the effects of manual traction, and when traction apparatus is properly applied the joint, supported and protected in Nature's indicated position and relieved from the constant traumatism of its own effort, begins to mend. The muscles, relieved of their duty of splinting the joint, relax their grasp. The deformity soon becomes modified, and after a few days or a few weeks the deformity is removed or sufficiently modified so that the patient can walk about in the open air and gain that great advantage which properly applied portative traction apparatus gives—viz., gentle exercise and locomotion in the open air, with a minimum of restraint as applied to the general functions of the body.

We may now formulate some of the conclusions applicable to the conditions found in hip disease, and its mechanical treatment.

1. The initial signs of the first stage of hip-joint disease are, as a rule, a limp and an involuntary muscular protection of the articulation.[1]

2. The involuntary muscular protection frequently, if not always, precedes the limp. It also precedes the pain, which is generally a very late symptom, occurring almost always in the second stage.

3. The more insidious the approach of the disease, the greater, as a rule, is the necessity for ample protection and prolonged observation. This is particularly true of an insidious and inveterate muscular protection of long duration which scarcely varies from month to month, and, which, while limiting the joint motions only slightly in all directions, is unaccompanied by any evidence of pain, except as the femur is used as a lever to overcome the muscular resistance.

4. The first indication from a standpoint of mechanical treatment is to remove as far as possible all traumatic influences from the diseased articulation.

5. This can best be accomplished by some form of apparatus which possesses the power to directly modify, or to wholly remove, interarticular pressure.

6. Our efforts to relieve the pain of the acute stage are abortive until a traction force is used.

7. The deformity of hip-joint disease is due to an instinctive and involuntary muscular splinting of the articulation, which muscular protection becomes modified after efficient traction is applied.

[Note by the author, September, 1923.]

[1] It was thought and hoped that the X-ray would aid in establishing primary bone changes in the early stage of hip-joint disease. Unfortunately this has proved fallacious.

8. The presence of the muscular splinting of hip disease necessarily indicates the existence of a greater or a less degree of interarticular pressure in those positions where muscular contraction resists the reduction of the deformity.

9. This muscular splinting exists very often to a very great degree without any pain at all, and in these cases, as well as in those where pain is present, traction is necessary to adequately protect the diseased articulation.

10. The muscular signs of hip disease, especially the instinctive and involuntary muscular protection, are by far the most important, both as applied to diagnosis and prognosis in hip disease, taking precedence over pain.

11. Clinical experience proves that all the important signs and symptoms of hip disease are at once modified by intelligently and scientifically applied traction, the modification of the symptoms and signs being in almost direct ratio to the efficiency of the protection thus afforded the affected articulation.

12. Where there is no deformity present, a portative traction apparatus may be applied, and the patient may at once assume the erect position (ample protection being afforded the diseased joint) and walk about in the open air almost without restraint.

13. If the disease permits a certain amount of motion at the affected articulation, motion within the limit set by Nature is not harmful. It is only the cases that Nature attempts to immobilize by an emphasized muscular splinting that require artificial immobilization, and these latter cases acquire motion under the immobilization produced by traction applied with this end in view.

14. Where deformity is present, the patient should be placed in the recumbent position, with the affected member at rest upon an inclined plane in a proper traction

apparatus. Both the traction apparatus and the inclined plane should be so constructed that any desired position may be obtained without disturbing the patient or the affected joint.

15. The deformity assumed by the involuntary muscular action indicates approximately the position in which we should make our initial mechanical traction.

16. Clinical experience proves that to make traction "in the line of the deformity" is an error. We should rather say that traction is to be made *in the line of comfort*, and this means that the angle of traction should be less than that of the deformity—*i.e.*, the joint should be placed in a position of greater flexion than that which is the exponent of the disease. By doing this we make traction wholly in a direction which modifies interarticular pressure, and we avoid the error of the lever apparatus, which makes a fulcrum of the diseased articulation to overcome the deformity.

17. The time necessary to overcome the deformity should always be given with the patient recumbent, as above described, and the line or angle of traction should be changed, not arbitrarily from day to day, but as the relaxation of the muscular splinting allows a change of position without any injury to the joint.

18. As soon as the deformity is reduced or sufficiently modified, the traction apparatus arranged for locomotion should be applied, and the patient should be allowed to have the benefit of gentle exercise in the open air, without interfering by crutches or other unnecessary apparatus with the normal functions of the rest of the body and the unaffected members.

19. Traction during recumbency with an apparatus that has a strong pelvic band and double perineal pads is superior to the weight-and-pulley traction, inasmuch as

the former gives us complete control over the pelvis as a point of counter-traction.

20. If the joint is very painful and sensitive—even if there be no deformity—the joint should be given the extra rest which recumbency, traction, and a relaxed ilio-psoas muscle bring. The presence of a cold abscess is not a contra-indication for locomotion in a proper portative traction apparatus, nor are several sinuses a bar to outdoor treatment with a proper protection splint. The only contra-indications to locomotion and outdoor life are a very sensitive joint, an acute phase of the abscess, and the existence of a considerable deformity.

21. Any apparatus embodying the lever principle alone —that form or those forms of apparatus which make the femur the long arm of a lever to "pry" the joint straight—are, I believe, very strongly contra-indicated, except in those cases where the articular adhesions prevent the reduction of the deformity by traction. Luckily, these cases are rare. When met with, however, I believe that it is better to reduce the deformity at once under ether, and then to apply efficient traction, rather than to subject the diseased joint to the prolonged and irritating traumatism of a powerful and constant leverage.

After reviewing the whole field, it seems to me that we can not avoid the conclusion that we should use traction as a means of relieving the self-imposed muscular traumatism of the joint, not only to meet the very evident general mechanical conditions, but to relieve the muscles as well. The muscles were long ago tired out before the painful stage appears, and we should appreciate the constant strain the muscular tension imposes upon the central nervous system. It has been taught that we should make traction to "antagonize the muscular spasm." I think this is an error. It seems rather that we should rest the joint by giving it immunity from the traumatism of interarticular pressure, and then await the gradual yielding of the muscles. This will soon permit us to place the distal part of the joint in the desired position, and we need not inflict the slightest injury to the joint if we proceed carefully. I am quite sure that this object can be secured by the intelligent use of traction and countertraction.

And the mechanism of this traction apparatus should be so arranged that it can be adjusted to any desired position without removing it from the patient. We can then cause the apparatus to follow the thigh as it falls into position and not force the thigh to follow the apparatus, as we must always do if we use the lever apparatus without the traction attachment.

The objections to any form of apparatus which embodies the principle of the lever alone are very great. It seems to me that this principle as applied to the treatment of tubercular joint disease in the light of the pathology of the disease, in the light of the muscular conditions which accompany it, and the unerring certainty with which the lever aggravates the already existing traumatism, is crude and unscientific.

Those who favor and use the lever apparatus always insist upon the necessity of removing the superincumbent weight of the body from the diseased joint. They forget or ignore the fact that the underlying articular pressure produced by the muscular splinting of the joint is just as injurious, if not more so, than the superincumbent weight. It is easy enough to remove the superincumbent weight with crutches (but this alone will not cure the disease), and it is also easy to gain position by making the femur the long arm of a lever. The muscles must yield if the lever force is sufficient, but the joint already disabled will suffer.

It will be interesting in the future to see the results produced by this lever plan of treatment.[1]

But if, on the other hand, we can successfully give the affected joint not a fictitious "rest," not a pseudo-"immobilization," not a fallacious "fixation," but by supplementing the artificial joint protection, be it what it may, with an additional apparatus or attachment which will imitate the action of manual traction, we shall accomplish the end that Nature seeks by her ceaseless and involuntary muscular protection is an osteitic joint disease. We must do more than "rest," "immobilization," or "fixation," as described by some writers who use these terms, can produce, because all these "methods" have for their aim, not so much the avoidance of traumatic contact between the opposing joint surfaces as the maintenance of a traumatic contact which already exists and which must necessarily be emphasized by the application of the lever principle or of any of the so-called "fixation" splints. As long as the muscles guarding a diseased hip-joint are in a state of ceaseless contraction, as long as they are actively and instinctively engaged in securing for the diseased articulation the best protection they can give, our mechanism for the relief of the joint should embody some means of successfully combating the effects of their incessant action. And we should always be prepared to produce true physiological rest for the over-tired muscles and the vulnerable articulation; we should be able to secure real immobilization of the joint when it is indicated, and we should always be ready to give the joint absolute fixation when it seems necessary, just as we should permit and even encourage motion when the joint condition does not contra-indicate it.

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[1] In "An Analysis of Twenty-one Cases of Hip Disease treated by the Thomas Splint," by Dr. John H. Huddleston (*Boston Medical and Surgical Journal*, Oct. 24, 1889), it was found (the Thomas splint being a typical "lever-fixation" splint) that the general results were as follows: "Good position with little flexion and adduction, but great shortening, great atrophy, and very constant elevation of the trochanter above Nélaton's line with a remarkable percentage of abscesses."

And, as I look over the entire armament of orthopædy, I know of no mechanical method of accomplishing all that we desire in these difficult and tedious cases of hip-joint disease except that which imitates the simplest method of relieving an inflamed joint—viz., manual traction.

And I feel that if this principle of traction be scientifically utilized and adapted to the needs of each individual case there is no doubt that we shall give to our patients suffering from hip-joint disease and allied articular affections the most perfect mechanical protection available, which is, after all, the *sine qua non* in the treatment of chronic joint disease.

WHAT ARE THE INDICATIONS FOR THE DISCONTINUANCE OF THE MECHANICAL TREATMENT OF HIP-JOINT DISEASE? WITH REMARKS ON SYMPTOMS AND TREATMENT.[1]

Read before the Orthopædic Section of the New York Academy of Medicine, May 15, 1891.

In the year 1873 there came under my care a little boy, aged seven years, who was suffering from hip-joint disease. The symptoms of the disease had been so slight and the invasion of the disease had been so insidious that several surgeons had denied the existence of a chronic hip-joint inflammation. But when the patient came under my observation, in consultation with the late Dr. Charles M. Allin and the late Dr. Henry B. Sands, there was no doubt as to the diagnosis. The patient was quickly put in a traction apparatus, and for several months he improved rapidly. The pain had disappeared, the slight adduction and flexion of the thigh had gone, and when the apparatus was temporarily removed the patient walked with a no greater limp than that which might be attributed by many to the continuous use of the apparatus for nearly a year. Aside from the limp, the only thing of importance which remained was the impaired motion of the articulation, which upon examination passed through only about half the movement of a normal hip-joint. This resistance to full and free joint movement was due wholly to a reflex muscular condition, as was proved by the administration of ether, under circumstances about to be related.

The father of the patient—a kind and sympathetic man—became impressed with the idea that the apparatus was injuring his child, although the condition of the patient had in every way improved under its use. He almost insisted that the splint should be removed, and, my objections and reasons not prevailing, I declined to remove the splint unless the proceeding was sanctioned by a formal consultation. To this the father willingly agreed, and a prominent surgeon was asked to examine the patient with me. A radical disagreement resulted. The consultant insisted upon the removal of the apparatus, a full return of exercise and liberty to the joint, and expressed himself as fully satisfied that the patient would suffer serious injury if the use of the apparatus was continued. My position was that a further use of the apparatus was necessary; that the joint, though not tender or sensitive to the conventional test of a pretty severe manual concussion, was still far from well; and finally that the reflex muscular conditions about the joint showed plainly that the articulation was far from being in a normal condition.

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[1] *The New York Medical Journal*, for November 29, 1891.

The father was informed of the result of the meeting, and at once suggested that a third surgeon should be added to the consultation. This was done, and, at the suggestion of the "referee," ether was administered. Under the influence of the anæsthesia thus produced there was no resistance to full and free joint movement in all directions. This condition, it was claimed by the newly added surgeon, proved that there was no true muscular protection to the joint; and he advised that the apparatus should be removed. In short, the writer was overruled in his position and views, although he insisted that the reflex character of the muscular resistance was the important diagnostic sign of hip-joint disease, and that the anæsthesia of ether would necessarily remove the purely reflex muscular conditions of hip disease, as well as, for example, the muscular conditions of hysteria.

Under protest, and with many misgivings, I removed the apparatus and watched the patient with great interest. The subsequent history of the patient may be briefly related. The limp became emphasized almost immediately, and the pain returned in a few weeks. Even then the father did not see the necessity of a return to the ease, the comfort, and the safety of the traction splint. It was not until the pain became very urgent and deformity reappeared that he consented to a renewal of the traction apparatus. The patient again improved after the apparatus was reapplied, but not so rapidly as during the period of its initial use. Before long a large abscess appeared, after which the patient steadily failed, and nearly three years after the first application of the apparatus he died as the result of prolonged and profuse suppuration. Excision of the joint was proposed after the suppuration became urgent, but the proposal was rejected by the friends of the patient.

In 1889—sixteen years after I first saw the patient above referred to—his cousin, having exactly the same name, came under my care for hip-joint disease. In this case, however, the invasion of the disease was somewhat different. The first stage was not so insidious as in his cousin's case. Still, the symptoms were very pronounced—the limp, pain, reflex spasm, and deformity were all present to a marked degree, and the outlook for the second patient was not at all promising. He was a very poorly nourished boy of six years, and his general condition was very unsatisfactory. The apparatus was duly applied, and for the first six months of treatment he received good attention at home and slowly but steadily improved. After that time the illness of his mother, plus some financial troubles, interfered with his visits to me. After about a year of treatment the mother made up her mind that mechanical treatment was no longer necessary, and she removed the apparatus. Three months later the mother brought the patient to me, and, with pardonable pride, showed me her child's hip.

The improvement was marked. The child walked without a limp. The muscles of the thigh were in excellent condition. The child's health had improved, and there was almost perfect joint motion. The reflex muscular spasm at the affected hip-joint was still present, however, though not in a marked degree. The muscular spasm was only slightly present in flexion, abduction, and adduction of the thigh. It was quite apparent in extension and in rotation inward during flexion. I presume many surgeons would have said the patient was free from hip-joint disease—and so he was, in a certain negative sense. But the joint was still vulnerable, as it is in the first apparent stage of the disease. It seemed quite probable to me, however, that a single major traumatism would reproduce the more urgent symptoms of the lesion. In order to insure the future permanency of the good result so far and so luckily obtained, I advised the mother to use a modified traction splint with limited motion at the knee for a few months longer, and the child, when I heard from him last, was wearing such an apparatus, and was slowly and surely improving.[1]

These two cases convey an important lesson; and they are cited from many others which might be related to demonstrate the difficulties that sometimes attend an answer to the question: "When are we justified in discontinuing the use of apparatus in the treatment of hip-joint disease?"

The first-named patient had the advantage of the best surgical opinion and advice in New York. Still, the result was very disastrous. In the second case the mother assumed the responsibility of removing the apparatus; yet the result was certainly very satisfactory. In the light of a more recent experience, I believe the first patient might have recovered if the traction treatment had not been interrupted, if it had been persisted in for a sufficient length of time, and if the traumatism inflicted by the ether test had been omitted. In the second case we have no means of iudging just what the joint conditions were when the apparatus was removed. Perhaps they were of a character that would have warranted the preliminary trial that we have so often advised. But when I saw the child I believed that he was not free from the danger of a relapse; and, as I knew that the further use of protective apparatus could do no harm, I advised its continuance as a matter of precaution and safety.

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[1] *November 17, 1891.*—I saw this patient a few days ago. The joint conditions had not improved with the modified traction splint. I regret now that I did not use the simple traction apparatus without motion at the knee-joint.

The indiscriminate use of the ether or chloroform test in hip-joint disease can not, I think, be too strongly deprecated. In my own experience, the administration of an anæsthetic as a test has been almost always followed by an increase in the more important symptoms, and the reason seems very plain. Anæsthesia removes Nature's only protection to the diseased articulation, "the reflex muscular spasm," and the opportunities for the infliction of traumatism to the joint when it is helpless are very great. It is certainly an unjustifiable proceeding when the joint has reached a stage where the question we are discussing to-night is raised. When we study the joint, we want Nature's expression of its condition. It is our safest guide. We lose all this when we administer an anæsthetic; and many a vulnerable joint, well on its way to recovery, has been hopelessly injured by the almost necessary traumatism of an examination under an anæsthetic. If we are obliged to use this test as a matter of *differential* diagnosis, our examination should be very gentle and our tests should be very carefully made; for one of the dangers of an examination under an anæsthetic is the breaking up of the encysted tubercular masses, thereby producing a fresh infection of the tubercular disease.

Recognizing long ago that tubercular joint disease—and this in hip disease means almost always tubercular *bone* disease—must necessarily run a very prolonged course; and realizing, even so long ago as the period of 1863 to 1868, when I saw so much of joint disease at the Hospital for the Ruptured and Crippled, that repair must necessarily be slow, I have long since ceased to expect that there was any "short cut" in the treatment of these conditions. It is true that we can, by efficient apparatus, place the joint under the best *local* conditions for repair; we can overcome deformity and hold the joint in the best position for use when repair finally comes; we can efficiently remove that most important factor in the progress of tubercular joint disease—viz., traumatism—and we can relieve the pain, procure normal sleep, and make our patients very happy and comfortable. We can add the very efficient element of activity and exercise to the means of cure, and make climatic influence a potent factor in assisting Nature to remove a tubercular disease. I think we are all agreed that tubercular joint disease is self-limiting, and that we may aid Nature in reaching the period of self-limitation by efficient mechanical treatment; and while I think that we can always hope to abort the disease in its first apparent stage by scientific orthopædic measures, the end that we all seek, after disintegration of the joint has commenced, is not controlled as much as we are apt to imagine by the mechanical means employed; for there is no apparatus and no "mechanical method" that will *cure* tubercular disease of the joints, just as there is no apparatus, for example, that will *cure* a fracture of the thigh.

After several years' experience in the New York Orthopædic Dispensary and Hospital, Dr. R. W. Lovett, then an assistant surgeon to the institution, kindly aided me in an attempt to learn the ultimate results of the mechanical treatment of hip-joint disease pursued in that institution. The result of our efforts has been published.[1] A brief reference to the basis of our investigation may be permitted here, as it illustrates some of the difficulties experienced in reaching a conclusion as to the question to be discussed by us to-night. In the paper above mentioned it was stated, among other limitations that controlled the investigation, that "no case was to be considered which had not been 'discharged cured' at least four years prior to the investigation," and "that many, if not all, of the patients treated in the dispensary are *experimentally* discharged as cured before they are finally entered as *actually* cured. To explain: A patient answering to the tests we apply would be permitted to go without his apparatus as a matter of experiment for a few weeks—weekly observations being made in the meantime. In some instances symptoms would reappear during this preliminary trial after an indefinite period. The apparatus would then be reapplied and experimentally removed again some months afterward, and this operation would be repeated until the final test of time proved that the recovery was permanent. The four years' limitation was introduced to cover the doubt that might arise regarding the permanency of the result."

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[1] "The Ultimate Results of the Mechanical Treatment of Hip-joint Disease." *New York Medical Journal*, May 21, 1887. See page 280.

It would seem that the four years' limit above mentioned ought to be sufficient to base a final report as to the results of treatment in almost any condition. But it is certain that in some cases of hip-joint disease even this period is not enough, for several of the patients reported as cured in the paper above referred to have relapsed during the four years since the report was made. In short, it is very difficult to say, in many cases, whether the disease is really arrested—just as it would seem certain that even if the tubercular process has become latent in some cases, there may yet remain a subinflammatory condition with encysted tubercular material at hand all ready to reinfect the vulnerable tissues upon the application of the proper traumatic influence. I have seen within the past month a supposed cure of hip-joint disease which relapsed sixteen years after the patient had been discharged, a slight traumatism being the only apparent cause of the relapse. We may sometimes imagine that our patients are cured, and years afterward discover that we have been mistaken—and we are very prone to forget some of our relapses as we recall, and perhaps speak of, those who have been cured.

What orthopædic surgery needs is more clearly ascertained pathological facts and less dogmatism as to methods of treatment; less enthusiastic adherence to special forms of apparatus and a broader interpretation of clinical data; less of theorizing and a more earnest search for the truth. Until this point is reached, it may safely be said that the real literature of orthopædic surgery will remain unwritten.

It is not necessary on this occasion to detail the symptoms of hip-joint disease. It does seem necessary, however, to call attention to the fact that many surgeons ignore the most important symptom of this condition. I refer to the principal neuro-muscular symptom—to the so-called reflex muscular spasm, which, if studied carefully, will tell us more about the joint condition than all the other signs and symptoms which characterize hip-joint inflammation. In the first case related above the surgeons called in consultation ignored the involuntary muscular protection of the joint, and, as the anæsthesia of ether abolished it, they regarded it of no importance. As in the early diagnosis of hip disease the signs and symptoms which are of real value are those of which the patient makes no real complaint, so, when the last stage of the disease is reached, and the question arises, "Can we safely discard mechanical treatment?" the answer will be decided almost wholly by the presence or absence of the involuntary expressions of disease, as shown by the attitude of the patient—the "joint sense"—the ability to use the affected articulation, and the general history of the case. In short, the question I ask myself under the circumstances is this: "Would I, if I saw this patient for the first time, apply an apparatus?" We are too apt to think that our patient is *cured* when he is only *better*, and, from our constant and frequent observation of the patient, to become careless in the treatment of such a chronic disease as the one under consideration. And if there is a true neuromuscular protection of the joint, there is no doubt regarding the necessity for continuing the support—if not to prevent disease, to prevent deformity.

If we could make the absence of pain a safe criterion as to whether or not a chronic tubercular process had ceased in a joint, our task would be an easy one. But we can not do so. The worst and most inveterate cases of hip-joint disease do not give evidence at all of an oral expression of pain. It frequently occurs in tubercular joint disease that the morbid process is so insidious, but nevertheless so profound, that there exists no expressive evidence of the disease outside of the deformity, the limp, the muscular atrophy, and the reflex muscular protection. Simple locomotion—or simple passive motion—becomes a traumatism under these circumstances. And until this is fully understood by the medical profession there will be unnecessary errors in diagnosis, and patients who might be saved from deformity and death will go unrelieved. On the other hand, there may be an abscess present or there may be sinuses which are discharging, both of which are no longer connected with the joint, and yet the articulation may be in a condition where mechanical treatment is wholly uncalled for.

The closing of sinuses leading to an abscess within the joint does not always indicate that mechanical treatment may be dispensed with. On many occasions I have looked forward to the healing of the sinuses as being the end of the disease, but clinical experience proves that in many cases the disease may change from the humid to the dry form of caries, and in these cases the change is always from bad to worse. My experience proves that the strictly speaking suppurative forms of hip-joint disease are those which run the most benign course, and that the more accurately the disease resembles an acute lesion, the better is the result, and the sooner the mechanical treatment may be discontinued.

There is one test which, negatively speaking, is of considerable value. A patient with hip disease who is unable, while in the supine position, to raise the affected member from the couch with the knee fully extended, is not in a condition to dispense with mechanical protection to the affected hip-joint. There is no more expressive sign of the inability of the joint to sustain the traumatism of locomotion than that shown by this test. There is nothing more positive, as applied to the inability of the muscles to obey the will which exists in hip disease with the joint unprotected, than that afforded by this test. As the joint conditions improve, the patient, who could not raise the straightened limb from the couch, will gradually regain this power, and this ability to raise the straightened limb is the first important sign of improvement in many cases. Still further, the patient who can not execute this movement without assistance can generally execute it readily if a traction splint is used with an ordinary amount of traction applied, proving that the muscles are competent to act if the interarticular pressure is relieved. It would seem during this test, with the traction applied, at first glance that the movement took place at the affected articulation. But this is not so. The traction apparatus so perfectly fixes or immobilizes the hip-joint that the pelvis and the femur become practically one bone, and the real motion occurs at the dorso-lumbar spine and at the opposite healthy joint. This test amply demonstrates the ability of the traction apparatus to immobilize the hip-joint. But the ability to flex the thigh upon the pelvis with the knee extended is not, unfortunately, a positive evidence of cure. It indicates improvement, and it is an encouraging sign of progress.

The ability of the joint to bear the conventional test of even severe normal concussion is not, unfortunately, an evidence of cure. Many patients with progressive disease, especially dry caries, stand this test without flinching at all.

And, finally, the absence of abscess proves nothing. The joint may be in a condition of slow but sure tubercular disintegration without any sign of abscess whatever. The worst and most obstinate cases of hip-joint disease are those which do not present any external evidence of suppuration.

There are, I believe, only two elements upon which we can positively rely in reaching a conclusion as to the propriety of discarding the mechanical treatment of hip-joint disease. These are: 1. The gait and attitude of the patient when locomotion is attempted without protection. 2. The character of the resistance to normal joint movements, as shown by the neuro-muscular protection, and the quality of the motion thus obtained.

In studying gait and attitude it becomes necessary to discriminate between the limp of chronic joint inflammation, the limp of a vulnerable articulation in the convalescent stage, and the limp of disuse and shortening.

The limp of the first condition is almost characteristic of the disease. It is marked by an expression of timidity

—an instinctive and involuntary refusal to bear the weight of the body upon the affected joint. The step is very greatly modified by the instinctive effort of the patient to avoid interarticular pressure; and, instead of taking a full, easy step the foot of the affected member oftentimes barely touches the floor, so quickly does the sound member come to its relief. Speaking mathematically, the affected joint does not do more than a small fraction of the work of locomotion. This limp may be modified by the stage or condition in which the examination is made. The instinctive protection of the joint is, however, present to a greater or less degree.

The second—that of vulnerable joint—is more like the limp, if I may be allowed the expression, of a unilateral fatigue. The joint has undergone repair to a certain extent. It may be that the tubercular process has practically ceased, or that the tubercular masses have become encysted. The patient has a sensitive joint, rather than an inflamed one, and, while there may be a conservatism about the joint movement in locomotion, the involuntary quality of the movement is lost. The joint is convalescent. It has reached a point where it can do a little work, but is not up to its full power of a normal resistance to traumatism. The step is not one of normal length, but approximates the normal in duration. It differs in all important respects from the true joint limp, and it may even exist with a modified tubercular process.

The third—the limp of disuse and shortening—may be described as the easy swing of a person who is simply lame, and who has simple muscular inertia and a slight mechanical impediment to locomotion. There is no expression of disease about it. There is no hesitation, no instinctive protection. The patient is simply lame.

The simple lameness of a cured hip disease is not difficult to recognize. It is oftentimes more difficult to differentiate between the limp of the true disease which has been somewhat modified by treatment and the limp of convalescence. The use of apparatus, too, may complicate the question, for, after an apparatus has been worn for several weeks or months, the knee does not at once resume its full function in motion, and this complicates the matter. But, if one is familiar with the expressive limp of true joint disease, he will have little trouble in deciding whether or not the limp is one that indicates a condition of joint inflammation. In doubtful cases it requires a prolonged study of limp, gait, and attitude to make them available in reaching a conclusion, and no positive conclusion can be reached unless the joint itself is carefully tested and its contribution to the general fund of information is added, for there are some patients who walk comparatively well, in the advanced stages of the disease, who need ample protection to the articulation—even as much as those who are lucky enough to have a slight pain, for pain is a symptom that is rarely ignored, even by those who have had but little experience in chronic joint disease.

No satisfactory conclusion can therefore be reached until the joint is carefully tested, and the most important symptom presented by the joint itself is that which I have ventured to call the neuro-muscular. The purely instinctive and involuntary character of this neuro-muscular sign makes it the most valuable aid we have in deciding upon the conditions existing in a tubercular hip-joint. The faithfulness with which the muscles obey the neural edict, the constancy with which they keep up their guard, and the persistency with which they resist, once the limit set by the nervous system is reached, appeals to me more and more as I study their earnest protest against traumatism. And this neuro-muscular element is always present in hip-joint disease, or, if it is absent, we are unable, as a rule, to make a diagnosis. We may know after treatment has commenced, if the muscles relax their grasp, that the joint conditions are improving; we may know, if the muscles increase their control of the joint, that the pathological condition is retrograding; and if we are assured that there is no instinctive muscular protection of the joint, there is little or no need for mechanical protection. If there were no neuro-muscular element present there would be no deformity, and the danger of recurrent adduction and flexion (the bane of relapsed hip-joint disease) would be removed; so that we may safely make the quality of the joint movement and the character of resistance to normal movement pretty safe guides in deciding the question we are discussing.

First, as to the *quality* of this joint motion. It is not usual to find any crepitation or roughness of the joint within the limit set by the muscular protection of the articulation. Even if the articular cartilage is eroded, the tubercular masses give us a smooth surface, upon which articular movement occurs, and the same condition is found when the pathologically altered femoral head plays against the diseased socket. The motion that we generally find is smooth and almost normal in quality, and if we discover true bony crepitation it is certain that the joint is far from being in a condition to bear the weight and pressure of locomotion.

Secondly, as to the character of the resistance to normal joint movement. That this resistance is due to a muscular check I have often proved by the ether test. I think all who listen to me to-night will admit that a true reflex muscular protection exists in tubercular osteitis of the hip. In advanced cases—when repair has commenced or when there are adhesions, either extracapsular or intracapsular, or both—the amount of movement under a profound anæsthesia may be far from the normal, but the reflex character of the resistance which exists during consciousness can be readily demonstrated. And in our examination of a tubercular hip-joint, while the patient is not under the influence of an anæsthetic, we may find the muscular conditions permit a great degree—almost a normal amount—of movement; or we may find the joint locked by an intense and unyielding contraction of all the muscles. In tubercular hip-joint disease this limitation of motion is due to a characteristic tetanoid spasm of the muscles, and we may know, if it is present even to a slight degree, that the joint is vulnerable and is not removed from the danger of a relapse.

We do not always see our patients with hip-joint disease in the first apparent stage before deformity has occurred, and where the limp and the neuro-muscular symptoms are our only guides to diagnosis. We are rather met by a larger class of cases where the disease has progressed to the point of deformity, with or without pain, and where we must fight the battle on the basis of a badly damaged joint. These are the cases that I desire to talk about to-night as applied to the question raised in this essay. The cases seen early, that have almost normal joint movement and that have no pain or deformity, are not usually the cases that puzzle or bother us, although there are cases which commence very insidiously that seem to defy any and all treatment. We can help Nature more in the ordinary incipient cases, because she needs only slight help. What I would like to describe, if I can, is, how we may know when it is safe to discontinue treatment in the cases that have reached the deformity stage before proper treatment was applied.

I think it proper to assume that we are all agreed that the conditions presenting in hip-joint disease demand mechanical treatment. We may differ somewhat as to methods; we may even, in a friendly way, disagree as to principles; but we are all striving after the same object—viz., the relief and cure of a distressing disease.

My experience has brought me many times in contact with the problems raised by the question we are to discuss to-night, and I think I may say that I am not altogether unfamiliar with the various mechanical methods that are employed in the treatment of hip-joint disease. And I feel that I should err if I did not call your attention to the superiority of the old Taylor splint—with its strong pelvic band and double perineal pads, and with its adhesive-plaster traction—over all other forms of apparatus in the treatment of doubtful cases during the period of convalescence. I have over and over again removed the straight traction splint when the joint symptoms became, as I thought, sufficiently modified to admit of a modified perineal support, and have used various instruments with limited knee-joint movement and without any mechanism to control the knee, and with and without adhesive plaster. I have sometimes found the limited knee-joint apparatus to answer, but rarely the apparatus with uncontrolled knee-joint movement. There is something peculiarly efficient in the old straight adhesive-plaster traction splint. It controls the element of traumatism, it prevents deformity, and it relieves the joint as no other apparatus does. Its superiority over any other form of apparatus, it seems to me, needs no demonstration; and its efficiency in removing traumatism from the joint and in producing a practical immobilization of the articulation are well-demonstrated clinical facts that need no indorsement from me.

The fact is that we are unable to immobilize the hip joint unless we control the knee and prevent its flexion. If the knee is flexed and extended even a little, motion must occur at the hip, unless the hip is ankylosed; and it seems not at all unlikely that the principal function of the Thomas splint is to prevent flexion of the knee. There has been a good deal said about the movement of the pelvis as a factor which prevents immobilization of the hip. It seems not at all unlikely that the knee-joint flexion is a more important element to study and control than the pelvic movement. This element of knee-joint flexion is readily controlled by the long traction splint, especially if efficient traction is kept up; but, just as soon as we give any liberty to the knee, we insure a certain amount of motion at the hip, and many patients in the convalescent stage can not tolerate this motion for a very long time. Not only that; when the ischiatic crutch splint is applied so that traction is efficient in the erect position, the perineal pads become loosened when the knee is flexed; and this is readily explained by the fact that the pelvic support is transferred, after the knee is flexed, from the tuberosity of the ischium to the ramus of the pubes which latter point is much nearer the knee when the thigh is flexed.

If, therefore, there is any serious doubt, keep up with the straight traction bar and the firm pelvic band with its two perineal pads. I have learned to think twice, or even three or four times, before I remove it, even in the convalescent stage of hip-joint disease; and my best results in cases where the disease has reached the deformity stage have been in those cases where this splint has been used the longest and the most continuously. For it seems demonstrated that the principal functions of any apparatus in the treatment of hip-joint disease are, first, to prevent traumatism; secondly, to remove deformity; and, thirdly, to maintain a correct position during convalescence; and these three factors are best met, in my judgment and experience, by the use of the long traction splint and the double perineal pads. There need be no fear of a prolonged use of this apparatus. It may increase the atrophy of the soft tissues, and it may exceptionally produce a greater bone shortening than would occur from simple disuse, but it does not produce interarticular pressure, as is the case with the Thomas splint and other forms of apparatus based upon the lever principle; and the malnutrition which results to muscle and bone is trivial when compared with that which results from a disease unnecessarily prolonged by a series of relapses. A straight limb, even if somewhat atrophied and an inch or so short, is a very useful member, as we all know.

It has occurred several times in my experience in dispensary practice that a patient with hip-joint disease would pass from observation for a long time—perhaps two or three years. But the mother or father, feeling that the child was safe in the splint, would keep up in their own way the principle of traction and protection. These patients have, some of them, returned to us with the tubercular process evidently entirely cured. Perhaps for five or six years they have worn the splint continuously, and perhaps not very efficiently; but, upon the removal of the splint, they would walk off without the instinctive limp of true joint inflammation, and with only the limp of disuse and a more or less shortened member. The muscles acted well; they were strong and efficient, and there was little or no deformity. They had been cured under a prolonged use of the apparatus—a prolonged use that might not have been sanctioned by the attending surgeon.

Prolonged treatment seems necessary in the majority of cases. Tubercular lesions of the joints are not easy to control, and repair takes place very slowly once the cartilage is gone and the bone is fairly attacked, or when there is a lesion at the epiphyseal junction, or at one of the centers of ossification. Recognizing these points fully, we may state that, if there is pain referable to the joint lesion, if manual concussion to the heel produces pain or flinching, if there is considerable deformity without ankylosis, if there is a true joint limp, or if there are abscesses or sinuses connected with the joint, we are not justified in discontinuing mechanical treatment. Or if there is reflex muscular spasm, limiting joint movement slightly in all directions; if there is almost perfect flexion of the joint, with the other movements considerably or markedly limited; if flexion and abduction and adduction are excellent, with rotation and extension limited; and, finally, if all the movements of the joint, except rotation inward during flexion, approximate the normal, there is almost a certainty that mechanical protection is necessary. Of course, it is understood that the limitation to motion above referred to is occasioned by the neuro-muscular protection peculiar to the disease. Not that it necessarily follows that an active tubercular process exists under all these circumstances; but, if too much liberty is given the joint under the circumstances above named, ordinary use of the joint becomes a traumatism, and a relapse, especially as to deformity, is almost sure to occur. This relapse may occur in a few weeks or months, or may be in a few years.

Rotation, and especially rotation inward during flexion, is always the last movement of the joint to recover. I have several patients now under observation who walk without a limp, who have no pain, and who have normal motion of the joint in every respect except rotation inward during flexion. Some of my patients have become married and have borne children, and they still have limited rotation inward during flexion; but there is no true muscular spasm in these cases. I doubt if they will ever recover this motion; and I believe this one movement may be impaired with an otherwise perfect integrity to the joint. Luckily, it is not an important movement, and its absence produces no inconvenience. But if rotation inward during flexion is limited by reflex muscular spasm, the joint is still vulnerable. The only safe thing to do under these circumstances is to remove traumatism from the articulation by some form of apparatus that acts independently of the patient's will, and which amply protects the joint without inflicting any traumatism by its own mechanism. The tubercular process, while slow and insidious, behaves kindly under scientific protection from traumatism; and one might write a long essay on the subject of the benign behavior of tubercular disease under non-traumatic treatment. This might be extended even to abscesses in tubercular joint and spinal disease, for the rarity of troublesome complications from abscesses, and the almost certainty of their absorption under efficient non-traumatic treatment, is becoming more and more evident to the writer every day.[1]

To return, however, to the subject under discussion. I have named some of the more important conditions which seem to forbid the discontinuance of mechanical treatment. Let us now consider the other side of the question and ask, What are the conditions which make it safe to remove the apparatus?

In answering this question much must depend upon the stage at which treatment was commenced and the general history and behavior of the disease under treatment. It is to be supposed that deformity has been overcome, that the

[1] See page 101.

question of abscess, etc., has been eliminated, and that the joint shows, by the test of attitude, limp, and gait, that the vulnerability has been so far modified that an experimental test can be made; for I feel that the first removal of the apparatus should be regarded as an experiment to be closely watched, with the chances of failure in excess. Bearing in mind the conditions which clearly contraindicate the discontinuance of mechanical support, to which reference has already been made, we are, as a final test, practically reduced to a consideration of the neuro-muscular symptoms. It does not answer to disregard the danger signal which Nature has kindly given us. I feel assured that, if a true neuro-muscular protection of the joint is present in the slightest degree, even if the limp is absent, there is danger. If there is only a slight degree of muscular protection, the danger is reduced to a minimum, and many patients, like the second case related in the first part of this paper, may recover almost full motion of the joint without prolonged mechanical treatment. But I am convinced that even in cases of this class some apparatus which modifies traumatism is necessary to secure the best attainable result; and I am very sure, from a prolonged and careful study of a large number of cases, that many patients in the deformity stage of the disease who are permitted to go about without support and who have true neuro-muscular symptoms will sooner or later relapse. It does not answer to rely upon negative evidence alone in these cases. It does not do to say: "There is only a very little reflex muscular resistance; it will probably wear away in time"; for the reverse is almost always true, and the little muscular resistance becomes a greater one in a short time. When the surgeon reaches a point where it would seem that the traction or some other form of apparatus can be dispensed with, there should be still kept up some kind of mechanical support which will modify traumatism for a prolonged period; and while the few cases which might recover without this intermediate treatment may wear the apparatus longer than seems necessary, the many will be benefited, and we shall have uniformly better results and fewer resulting deformities; for a vulnerable joint in the period of convalescence is pretty sure to become deformed, and an active tubercular process may be re-excited by a major traumatism or a succession of minor ones; and if some one could only invent the ideal intermediate splint, which would protect the joint with a minimum amount of inconvenience and expense, he would confer a great blessing upon orthopædic surgery.

I do not think any one exists to-day who can safely predict what course a vulnerable joint in the stage of convalescence will pursue under the removal of the restraint imposed by properly applied apparatus—and I think this is due to our very indifferent knowledge of tubercular joint lesions undergoing repair, to our lack of clinical signs by which we can know the exact location of the lesion, and hence judge approximately as to the effect of traumatism upon it. One can readily see that the lesion may be located at the joint surfaces or at a point contiguous to the joint, so that traumatism may affect it seriously or even only slightly. Hence we must be driven to an experimental trial and test, standing ready, by an early recognition of the signs which accompany or precede a relapse, to maintain the advantage gained by a prolonged treatment; and we must learn that we need not fear the effect of prolonged mechanical treatment upon the parts remote from the joint as much as we must dread the effect of an unheeded cry of the joint for prolonged protection.

A relapse may occur even after the limp has entirely disappeared. I have known this to happen several times.

In all these cases, however, the reflex muscular spasm persisted to a greater or less extent. A recent case illustrating this point occurred in the service of Dr. Myers at St. Luke's Hospital. After the usual treatment the hip-joint symptoms became very much modified and mechanical treatment was discontinued as a preliminary trial. The patient soon lost the "splint motion," and she could walk with a perfectly normal gait. There was no limp or any perceptible difference in the use of the two limbs in walking or running. The patient could even hop with the affected member with perfect ease. There was no pain or swelling or deformity. There remained, however, a slight but easily demonstrated reflex muscular resistance to rotation inward during flexion and to extension of the thigh beyond the horizontal plane of the body. The patient seemed so well that the father desired to remove her to her home in Massachusetts. I advised Dr. Myers, who kindly asked me to examine the patient to keep her under observation, saying that I believed that, if she went to her home without proper mechanical support, she would relapse. The father took his daughter home, against Dr. Myer's advice, and without a splint. After an absence of about six months she returned with the disease in the deformity stage, with marked reflex muscular spasm in all directions, with pain, a slight swelling in the inguinal fossa, and with the thigh markedly abducted, flexed, and rotated out.

This and the second case related in this paper, and other similar cases, make it seem certain that there is a recognizable stage of hip-joint disease which antedates the limping stage, and that the neuro-muscular signs precede the limp, as I have pointed out on a previous occasion.[1]

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[1] "On the Principles of the Mechanical Treatment of Hip-joint Disease." *N. Y. Medical Journal*, November 23, 1889.

The refuge of the surgeon in a condition so chronic, so difficult to relieve, and at times so uncertain in its result, would naturally be in some radical procedure. And surgery has not neglected the operative side of the question. Excision of the hip-joint has been performed many times, and its results have been before us for study and comparison for years. We may fairly reach the conclusion that this operation is not so popular or so frequently resorted to as it was a few years ago. Its results are far from satisfactory in many cases to which it would seem especially applicable. And this may be due, in a measure, to the fact already pointed out—viz., the lack of clinical signs by which we may recognize the exact location and the extent of the tubercular lesion. We do not know, when we cut, just what we are going to find. If we could be assured, for example, that there was little or no chronic osteomyelitis of the femur, or that there was no disease of the acetabulum, and the operation was limited to cases of this class, the results of hip excision would compare favorably, for example, with those of excision of the knee. But we are unable to determine, in our present state of knowledge, by clinical signs, the location and the extent of the lesion. In addition to this, it is almost impossible to completely excise the acetabular half of the joint. Deeply buried in the pelvis, and with only a very thin bony partition separating the joint from the pelvic fossa, we must in many cases perforate the non-articular portion of the acetabulum if we excise any considerable osseous tissue. The so-called operation of excision of the hip joint is generally a decapitation of the femur, coupled with an incomplete or imperfect removal of the tubercular disease at the acetabulum—in short, it is not a true excision of the joint. And the frequent reappearance of disease and the danger of prolonged suppuration after excision of the hip make it apparent that the operation is not always a safe or certain one in its results.

Notwithstanding all this, the operation is sometimes demanded by the circumstances surrounding the patient and by the expressed pathological conditions present, but I think it requires a very extensive experience and a more than ordinarily judicial medical mind to indicate the cases that would do well under an excision of the hip-joint.

On the other hand, the mechanical treatment, though often prolonged and generally tedious, promises better results to both life and limb than the operative measure. The conservative method may be more difficult, it may require prolonged training to make it successful, and our knowledge of the ideal mechanical treatment may yet be in its infancy; but I am assured that Nature aided in these cases is better than Nature forced, and my experience proves that it is better to await the self-limitation of the disease under proper mechanical, constitutional, and climatic treatment, than to attempt the radical removal of a tubercular disease in an articulation where Nature has placed so many barriers in the way of a complete and satisfactory excision of the entire joint.

The best results are undoubtedly obtained under mechanical treatment in those cases where the condition is recognized and treatment is commenced in the first apparent stage of the disease—where we have only slight neuro-muscular signs and a slight limp as exponents of the disease. In many, but not all, of these cases we may expect to secure perfect and free joint motion without any modification of rotation inward during flexion. After deformity and pain appear, and the neuro-muscular signs and the limp become pronounced, we may safely conclude that the integrity of the joint is seriously impaired; and we must work to secure the best attainable result, which may be a shortened member in good position, with or without movement at the articulation. And if someone among us will point out how we may secure early ankylosis in many cases of this class, he will earn a brilliant reputation.

My answer to the question which heads this essay is this:

In the first apparent stage of tubercular disease of the hip-joint—where there is no deformity present, and where we have only the neuro-muscular signs or the slight limp, or both, to guide us, as well as in the more severe stage of the disease, where deformity is present, where tubercular disintegration of the joint has commenced, and where the muscular protection of the articulation is more pronounced—the only safe guides for discontinuing mechanical treatment are, first, the absence of the expressive attitude and gait of tubercular osteitis of the hip-joint; and, secondly, an essential modification or an abolition of the instinctive neuro-muscular protection of the articulation. And, further, I believe that in all but the exceptional cases a relapse as to the deformity, or the disease, or both, is likely to occur as the result of the traumatism of ordinary locomotion, unless proper mechanical protection is maintained until the articulation is free from true reflex muscular spasm or is ankylosed.

ON THE ULTIMATE RESULTS OF THE MECHANICAL TREATMENT OF HIP-JOINT DISEASE

IN COLLABORATION WITH DR. ROBERT W. LOVETT, OF BOSTON, MASSACHUSETTS.

An Analysis of Fifty-one Cases occurring in the Service of the New York Orthopædic Dispensary and Hospital.[1]

Read before the New York Academy of Medicine, May 5, 1887.

THE attention of the medical profession of late years has been largely directed to the operative treatment of hip-joint disease, and the results obtained by excision of the head of the femur have been quite freely discussed. The more conservative method of mechanical treatment has not, during this period, received the attention it deserves, and it is the object of this paper to present for the consideration of the profession certain facts which have been developed by a thorough and painstaking analysis of the results which have been obtained by the treatment pursued in the New York Orthopædic Dispensary and Hospital.

The cases upon which the following report is made are drawn from the dispensary class only; private cases and those from hospital or other sources, as well as those in which excisions have been performed, being eliminated from the list, the principal idea of the writers being to arrive at the *ultimate result* of the strictly mechanical treatment of the disease, and to present an analysis only of those in which the patients were the recipients of dispensary treatment.

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[1] *The New York Medical Journal*, May 21, 1887.

Those who are familiar with the difficulties attending the medical or surgical control of an ordinary dispensary patient will at once appreciate the great difficulty of securing efficient home co-operation among the tenement-house class during the prolonged treatment of a typically chronic disease. And this is especially true when the treatment covers years, and when the successful issue of a case depends upon the intelligent use of an apparatus which requires constant attention. It is, however, under these circumstances that the treatment of the cases about to be reported was conducted, and some allowance as to the time consumed, etc., should be made. That the results are better in private practice, and sooner attained, no one, I think, will deny. It affords the senior writer of this paper great pleasure to record the fact, however, that the results reported were obtained under all the difficulties attending a dispensary practice among the poor, and he wishes, in this connection, to extend his thanks to the trustees of the institution for their generous co-operation and sympathy.

Indeed, it is among the objects of this paper to demonstrate that it is not only possible, but comparatively easy to treat such a serious condition as hip-joint disease successfully, and even satisfactorily, with proper dispensary facilities. The latter include, of course, a well-equipped shop and a corps of mechanics to make, and to repair or alter, under the direction of a trained orthopædic surgeon, the necessary appliances, and the co-operation of one or more out-door visiting surgeons. Without these aids, we feel assured the results obtained would not have been so satisfactory as those we have to report. Another object of this paper is to show that it is the exceptional case, that of the homeless child, or the child, with utterly indifferent or very poor parents, which requires *prolonged* hospital care in hip-joint disease. What the senior writer has frequently called "home treatment" enters largely into orthopædic dispensary work, and this means that an intelligent parent or an interested relative can, under proper instruction, carry out at home, in the great majority of cases, the directions of the surgeon, with occasional or periodical professional supervision.

There are, however, many poor children whose surroundings make hospital care necessary, as has been proved many times in our experience. But a well-equipped orthopædic dispensary, if properly conducted, can do the work of several large hospitals at a far less cost.

In the spring of 1886 the senior author of this paper requested Dr. Robert W. Lovett, of Boston, then an assistant surgeon of the institution, to personally investigate and to carefully record the *ultimate results* obtained by the conservative mechanical treatment of hip-joint disease practiced in the New York Orthopædic Dispensary and Hospital.

In order to arrive at positive and *ultimate* results, the following bases of investigation were formulated:

I. No case was to be considered which had not been under the care of the institution from the time of its entry to its final discharge.

II. No case was to be considered which had not been formally entered as "discharged cured" after a final examination.

III. No case was to be considered which had not been "discharged cured" at least four years prior to the investigation.

IV. No cases were to be considered except those which presented unmistakable signs of the disease at the time of the first examination.

V. None were to be considered which had not been at least two years under treatment.

VI. None were to be considered which were not seen or examined after the investigation was commenced.[1]

VII. All cases coming within these exclusive conditions were to be classified and reported.

While these exclusive conditions eliminate very many interesting and valuable cases which might have been legitimately added to the list, the writers of this paper believed that the facts brought out under the conditions named would have a positive value, and subject the methods pursued to more than an ordinary test. A much more brilliant, but perhaps not so scientifically accurate, statement might be made if the analysis covered *all* of the many other cases of hip-joint disease in which the patients had been discharged as cured, or if the time had been made six months instead of four years. It seems to the writers that the above-mentioned conditions must satisfy even the most exacting critic.

In order to further demonstrate the exclusive conditions under which this report is made and to arrive at approximate conclusions as to the percentage of deaths, we may state that for five of the years included in the seven from which the cases here reported are drawn—in other words, from 1877 to 1882—778 patients with hip-joint disease were treated. During these five years, 168, or 21.59 per cent., were discharged cured, and 50, or 6.4 per cent., died from conditions associated with or depending upon the chronic hip-joint lesion. The causes of death are as follows: Tubercular meningitis, 20; amyloid kidney, 5; phthisis, 3; exhaustion, 3; tubercular peritonitis, septicæmia, and convulsions, each 1; unknown, 16. These statistics regarding death are approximately correct, as can be vouched for by the assistant surgeons who have prepared the reports of the institution.[1] Deaths from the ordinary diseases of childhood are not included, and, though some of the unknown sixteen were doubtless due to some intercurrent acute lesion, they have all been included among those dependent upon the joint lesion.

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[1] In two instances the final record was made in the dispensary just before this investigation was commenced.

It may be stated that many, if not all, of the patients treated in the dispensary are *experimentally* discharged as cured before they are finally entered as *actually* cured. To explain: A patient answering to the tests we apply would be permitted to go without his apparatus as a matter of experiment for a few weeks, weekly observations being made in the meantime. In some instances symptoms would reappear during this preliminary trial after an indefinite period. The apparatus would then be reapplied, and experimentally removed again some months afterward, and this operation would be repeated until the final test of time proved that the recovery was permanent. The four years' limitation was introduced to cover the doubt that might arise regarding the permanency of the result, and all included in this report have been discharged as *actually* cured. The length of time some of the patients have been under treatment can be accounted for if these facts are duly considered.

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[1] Dr. Samuel Ketch, one of the senior assistant surgeons of the Dispensary, who, with the others mentioned, has had much to do with the preparation of the reports of the institution, furnishes the authors of the paper with the following information: In addition to the 168 discharged cured and the 64 who died (50 deaths only being due to the hip lesion) during the five years specified, 261 were "discharged relieved," 49 were "discharged for neglect," and 6 were "discharged as incurable." The 261 "discharged relieved" include all who were personally known by some member of the staff to be in a fair way to recover, the patients having, on account of change of residence, removal from the city, or some other cause, passed from under observation. The 49 "discharged for neglect" represent those who, after repeated trials, had failed to follow instructions. The 6 "discharged as incurable" comprise a small class for whom the opportunities of a favorable result were not possible in dispensary practice, and who could not be admitted into the hospital. They were not discharged on account of the pathological conditions present, for many apparently desperate cases have been carried to a successful issue by mechanical means alone. It was largely a question of deformity, not of disease, which gave rise to this classification.

The general plan of treatment may be briefly described.

In each case reported, a long Taylor traction-splint was applied soon after the first examination, and the parents or some friends of the patient were instructed in the use of the apparatus. The patient, unless recumbency was necessary to overcome a malposition of the limb, or unless the symptoms were so acute as to demand rest (in which case the patient was visited at home by the out-door visiting surgeon), was allowed almost unlimited exercise in the open air. He was instructed to call at the dispensary every week or two for observation and a readjustment of the apparatus, etc. If the patient's condition required recumbency for a month or more, and his home care was inadequate, he was placed in one of the wards of the institution, but was removed and placed in the dispensary service again as soon as walking was practicable.

The aims of treatment may be briefly summarized:

1. To overcome by mechanical means any acquired deformity that existed before treatment was commenced.
2. To protect the diseased joint from traumatism.
3. To permit the patient to have almost unrestricted outdoor exercise, and
4. To maintain that position of the limb which would reduce deformity to the minimum if ankylosis occurred.

In short, the results here recorded were obtained by simple conservative measures, the protection afforded the diseased articulation by the apparatus being the principal feature of treatment. In no case was any operative measure employed, except the occasional opening of an abscess, with, in some cases, full antiseptic precautions; in others by simple incision and ordinary dressing. But abscesses were not interfered with, as a rule, unless there were positive indications for the employment of the knife. And, as a matter of experience, abscesses connected with a suppurating hip-joint did no better under antiseptic measures than those opened by simple incision, and neither did so well as those which were allowed to open spontaneously. "Cold abscesses" were allowed to take their own course, unless they were in a location which interfered with the use of joint protection. Little or no medication was employed.

But these patients were as carefully watched as the exigencies of dispensary practice would allow. If they failed to report to the dispensary as regularly as directed, they were written to and requested to call, or in many instances they were visited by some one of the assistant medical staff. Indeed, although these patients were wholly from the poorer classes, many of them living in the ordinary tenement-houses, and had in very many instances very bad attention at home, they were cared for under the judicious management of the trustees of the institution by a very earnest and thorough corps of assistant surgeons.

And to these assistant surgeons much credit is due. When it is said that among them may be mentioned Dr. Samuel Ketch, Dr. John F. Ridlon, Dr. T. L. Stedman, Dr. Henry W. Berg, Dr. E. D. Simpson, Dr. Leroy W. Hubbard, and the late Dr. Simeon A. Foster, of this city, Dr. A. Sydney Roberts, of Philadelphia, and Dr. George B. Packard, of Hartford, the character of the work performed needs no further comment.

As affecting the question of result, it may be stated that the long splint, without any joint at the knee, was used from the beginning to the end of mechanical treatment. In private practice, an intermediate splint allowing either hip- or knee-joint motion, or both, is almost always used during the period of vulnerability of the joint, after the active symptoms have subsided. This intermediate splint is expensive, and for that reason not available in dispensary practice. It is altogether probable that better results, as regards both ultimate position and motion, would have been recorded in many cases had this intermediate apparatus been employed.

Before giving the results of the investigation, it may be well to say that it was first proposed that a detailed statement of each case should be made in this paper before giving an analysis. But the length of an essay presenting this amount of detail would be such that it would be impossible to read it in one evening. Hence it is that our conclusions only are recorded, but only after a patient and very exhaustive examination.

Of the patients with hip-joint disease who were under the care of the institution, and who were discharged as cured from 1875 to 1882, there were found among the number which were available under the exclusive conditions of this investigation 51 which could be traced. Many had disappeared, and prolonged and diligent search failed to find them. In the years that had elapsed they had moved to other places, leaving no trail by which they could be followed. A considerable number were inaccessible.

Of the 51 patients traced, 4 had died—2 of meningitis, probably tubercular, 1 four months, the other six years after discharge, and 2 of pneumonia, seven and ten years respectively after discharge, having been in robust health up to the time of their last illness. Six had relapses. They had been under treatment 2½, 4, 4, 4, 5, and 7 years respectively. In 1 case the apparatus was removed without the knowledge of the surgeon, but in the 5 others treatment was discontinued only after the joint symptoms had ceased for some months, and the patient could hop, run, and perform other active movements with the affected limb. One of these (the one whose splint was removed at home) remained well two years; 2 were well three years, 2 four years, and 1 five years before relapse occurred. In 1 case relapse occurred from a severe fall from a cart, but in the other cases no cause could be ascertained. As to the progress of these 6 patients with relapse, 1 had excision of the hip performed at one of the hospitals, and was still in bed after the lapse of one year, 2 are still wearing traction-splints and are nearly well a second time, 1 is on crutches and is slowly improving, and the remaining 2 have been cured a second time by the traction apparatus. The remaining 41 patients were seen at their homes and were carefully examined, with the exception of 2, who were reported to be in excellent health, and walking well at the end of four and six years, respectively. The remaining 39 were carefully examined and measured. These 39 patients had been discharged as cured during the period mentioned (from 1875 to 1882), and had been under treatment as follows:

TABLE I.

Showing length of time under treament.

2 years	4 cases.	6 years	1 case.
2½ years	4 "	6½ years	1 "
3 years	9 "	7 years	1 "
3½ years	6 "	8 years	1 "
4 years	8 "	Total	39 cases
4½ years	2 "		
5 years	2 "		

When these 39 patients presented for their first examination, the disease had been in progress, as stated by the parents or friends accompanying the patient, as follows:

TABLE II.

Showing length of time disease had existed prior to the commencement of treatment.

Length of time.	Cases.
Less than 6 months	8[2]
Six months to 1 year[1]	18[3]
2 years	3
3 years	5
6 years	1
7 years	1
9 years	1
For an indefinite time	2
Total	39

It can readily be appreciated, from studying the foregoing tables, that the cases reported are unselected. They represent all phases and conditions of hip-joint disease such as are likely to present in every-day dispensary work.

Of the 39, 27 had one or more abscesses at some stage of the disease, and 12 had none. But the latter were by no means the less marked cases or the ones followed by the best results.

Although, in the foot-note above, an attempt has been made to express in degree the results obtained in certain cases, the difficulty of conveying a definite idea as to results in this manner leads us to state definitely, in tabular form, when possible, the actual results obtained.

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[1] Of those applying with a history covering less than one year 19 gave a definite time. Two weeks, 2 cases; 4 weeks, 1; 8 weeks, 2; 4 months, 2; 5 months, 1; 6 months, 5; 7 months, 2; 8 months, 2; 9 months, 1; 10 months, 1.

[2] Of the 8 cases where disease had existed less than 6 months, the ultimate result was as follows: Perfect, 2; excellent, 2; good, 1; fair, 1; poor, 1; indifferent, 1.

[3] Of the 18 cases where disease had lasted from 6 months to one year, the ultimate results were: Excellent, 1; good, 4; fair, 11; poor, 2.

First, we will consider the difference in the length of the lower extremities.

The difference in the length of the legs, measured from the anterior superior spine of the ilium to the inner malleolus, was, when any difference existed, from half an inch to two inches and a half, with two exceptions. One patient, with dislocation of the head of the femur, had six inches shortening, and one (without abscess) had three inches. Two had absolutely no shortening. The amount of shortening did not seem to depend upon the presence or absence of abscess. The case with six inches shortening and dislocation ran its entire course without evidences of suppuration, while, on the other hand, the patients in whom there was absolutely no shortening each had abscesses. One of these children with no shortening had six distinct sinuses, the other two. It will be seen from a study of the following table, however, that the cases where abscess was present show more shortening than the others:

TABLE III.

Showing the relation of the presence or absence of abscess to shortening of the limb.

Shortening, in inches.	Cases with abscess.	Cases without abscess.
0	2	..
½	1	1
1	5	5
1½	4	3
2	8	1
2½	5	..
3	..	1
6	..	1
Total	25	12

The influence of age upon the ultimate amount of shortening is worthy of study.

In reaching conclusions on the point, the cases were divided into two classes:

1. Those in which the disease ran its course, and was cured before the age of ten was reached; and
2. Those in which the disease began at, or continued beyond, the age of ten years.

Table No. IV. has been prepared to show the effect of early and late disease.

TABLE IV.

Cases cured before 10.	Cases cured after 10.	Shortening in inches.
2	..	0
1	1	½
6	4	1
5	2	1½
3	6	2
—	5	2½
1	..	3
—	1	6

Eighteen cases cured before ten years represent an aggregate of twenty-five inches of shortening, while nineteen cured after ten represent thirty-eight inches of shortening.

In studying the general effect of shortening, it was found that it existed principally in the femur, though the tibia shared in it to a lesser extent also. The shortening of the thigh was ordinarily about two-thirds of the whole, but sometimes it was less, and in one case of two inches and a half shortening it was wholly in the femur. In two cases where there was a considerable shortening of both femur and tibia, the fibula seemed much less affected than the other bones. When there is much shortening of the leg, the foot of the affected side is also smaller than the other.

The difference in the length of the legs almost always increases slightly after the disease is cured. At the time of discharge from the dispensary, careful measurements were made and recorded in twenty cases. At that time the shortening varied from half an inch to an inch and a half, and in only four cases was it as much as two inches. Several years afterward these cases showed, almost without exception, an increase in the amount of shortening. In five cases it was an inch or less, and the rest showed from one and a half to two and a half inches difference. The legs had grown meantime five, ten, or even fifteen inches, so that the shortening was not in any case excessive. It also seems that the shortening does not increase indefinitely, for, in the cases where eight or ten years had elapsed between the two measurements, there was no greater shortening than when three, four, or six years had elapsed.

The difference in the circumference of the thighs, measured at the middle, was from one to three inches. In one case it was four inches, but the majority were between one and two inches. No constant increase or decrease took place as the years went by, some cases showing more atrophy than five years before, and some showing just the same comparative difference. The wasting, however, did not disappear, and in no case, even when the restoration of the joint was perfect, was the difference in the circumference of the two thighs less than one inch. Nor did the amount of atrophy at the time bear in the history of the disease any constant relation to the amount of shortening. It would appear that there may be a maximum of muscular atrophy with a minimum of bone shortening, and *vice versa.*

The atrophy of the muscles of the calf behaved in much the same way, except that it grew less in the course of years much oftener than the thigh atrophy did. It ranged from an inch to an inch and a half, and was ordinarily one half or one third as much as the atrophy of the thigh. In a word, it is apparent that the nutrition of the bones and muscles of the entire limb receives in hip-joint disease a shock from which it never entirely recovers.

No treatment was used to develop these atrophied muscles. The use of electricity, massage, etc., would probably have resulted in much better muscular development, as has often been the case in private practice.

Nineteen cases were found with practically ankylosed joints, six with slight motion in flexion, seven with motion in flexion of from 10° to 45°, and a certain amount of motion in other directions; three with motion to a right angle in flexion, and good rotation and abduction, and three were found with *perfectly free motion in every direction.*

It is very interesting to see from the subjoined table that the presence or absence of abscess seems to have but little influence upon the amount of motion after final recovery from the disease.

TABLE V.

Showing the influence of the presence or absence of abscess upon joint-motion.

Condition of joint as regards motion.	One or more abscesses.	No abscess.	Total
	Cases.	*Cases.*	*Cases.*
No motion in joint . . .	12	4	16
Slight motion	4	2	6
10° to 45° of motion . .	5	2	7
90° of motion	3	. .	3
Perfectly free motion .	2	1	3

Examination proved that the amount of motion grows less as time goes by. At the time of discharge several of these patients had a certain recorded degree of motion of the affected articulation. This motion had entirely disappeared a few years later, and in this table these cases figure as cases of ankylosis. Two patients in whom 90° of flexion was possible six years ago, had only 30° when examined in 1886. In no case was it noted that joint motion had increased since the final discharge and record. It almost invariably grew less.

An important practical point, as regards the use of the limb and locomotion in cases where ankylosis takes place, is the position in which ankylosis occurs. In 15 of the cases where there was little or no motion at the articulation there was no flexion of the thigh. The limp in these cases was trivial. In other cases the thigh was flexed at an angle of 120° to 135° with the horizontal plane of the body. This was not a serious impediment to locomotion when a flexible dorso-lumbar spine and a sound hip-joint on the opposite side existed, but in one case there was caries of the lumbar spine, and in this case there was very difficult locomotion. Flexion to 135° with a normal spine does not produce either difficult locomotion or a bad gait, and in no case examined did the permanent flexion exceed this angle.

Permanent *adduction* is a more important matter. Of 24 cases it was very slight or absent in 13, in 8 it equaled 10° to 15°, and in 3 cases it was about 30°. In two cases there was slight abduction of the thigh, and in one of these there was a condition of hyperextension of the knee. But even in this condition the patient walked well. In cases where abduction was present in the earlier history of the disease, adduction was found in the late history; and adduction is likely to occur after the removal of the splint, and to increase up to a certain point. But, as shown above, adduction to 30° occurred in only three cases, and in only one of these was it troublesome. In this case the slight flexion, with adduction to 30°, a real shortening of one inch and a half became, for the practical purpose of locomotion, a shortening of four inches.

It is desirable to maintain the parallelism of the limbs if possible. If the patient can place the limb squarely under the pelvis, locomotion is comparatively easy, even if flexion is present, and in cases where it is possible a protective, intermediate, perineal support with joints at the hip, knee, and ankle should be worn to prevent the adduction which is very apt to occur after the removal of the unjointed traction apparatus.

When the hip-joint is stiff the dorso-lumbar spine and the unaffected hip-joint have to do the work ordinarily performed by the two normal hip-joints. After prolonged walks or over-exertion in locomotion it was common for those examined to complain of backache. But this was only temporary, and disappeared after rest.

One or two were subject to slight attacks of pain in the hip in damp weather, but there was no sign of disease, and a relapse was not anticipated. Their health seemed perfect. Pain and weakness of the ankle on the affected side was common, and non-deforming club-foot or an inability to flex the foot beyond a right angle was present in twenty cases and absent in thirteen. The rest were not noted. It was, of course, most apt to be present when real or practical shortening made it necessary to reach down with the toes at every step. One patient was in a condition of acquired talipes equinus.

A very careful examination was made as to the presence of lateral curvature of the spine in nearly every case. There were some forty individuals examined, all of whom had some difference in the length of their lower extremities.

The difference varied, as above noted, from half an inch to six inches. They had, nearly all of them, this difference when they entered upon puberty. The conditions are those that we have been taught to believe are most potent in the production of true lateral curvature. Habitual malposition of the spine in standing, walking, or even in sitting was present in many cases. And yet in only one case of the whole forty was there anything that approached true lateral curvature. One girl, aged fourteen, had a slight but distinct lateral curve in the dorsal region, and a still smaller curve in the lumbar region. There was slight rotation of the vertebræ, as shown by the position of the ribs, when extreme anterior flexion of the spine was executed. The lateral curve was almost wholly obliterated when the patient was placed on her face and the pelvis was straightened, but the rotation persisted in a slight degree. There was no lateral rigidity of the spine. She had two inches true shortening of the left leg, which was practically three inches in the standing position on account of 15° adduction and slight flexion. There was in this case an incidental curve of the spine due to the tilted pelvis, but in many respects the curvature lacked the characteristics of true rotary scoliosis.

It appears from our investigations that the amount of shortening of the leg is not dependent on the presence or absence of abscess, that it is less in children who are cured before ten years, and that it apparently increases after that age is passed. It also appears that shortening increases slightly after the joint disease is arrested, which seems to prove that the affected limb did not grow so fast as its mate.

Muscular atrophy is always present in hip-joint disease. It persists after the disease is arrested and the apparatus is removed, and it may often improve after treatment is stopped, but never wholly disappears.

The amount of motion in the joint when the disease is arrested and the apparatus is removed is very apt to diminish somewhat in the course of years; but recovery with perfect motion is not impossible, or indeed unlikely. The presence or absence of abscess has no apparent effect on the amount of motion obtained.

Ankylosis being the result most likely to occur, it is important to study the ultimate position of the limb, so that, if ankylosis occurs, the most useful position will result.

Adduction, rather than flexion, is the ultimate position to be avoided, and adduction is apt to occur after the joint seems to be free from inflammation and the apparatus is removed.

Non-deforming club-foot, or even a considerable degree of acquired talipes equinus, may occur when there is much shortening of the leg with flexion of the thigh.

True lateral curvature with rotation occurs very exceptionally, if at all, as the result of even great inequality in the length of the lower extremities, only one case being found in the series, and that not a true rotation curve.

A comparison of the results here reported with the published results of excision of the head of the femur leads the authors of this paper to the conclusion that the conservative methods of treatment here described promise much better ultimate results than excision of the joint, aside from the greater mortality attending the operation.

We may briefly summarize the results of our investigations as follows: Of 51 patients discharged as cured over four years ago, 4 have died, 6 have had relapse, and 41 have apparently been cured of the disease. Of the 4 who died, 2 only had presumably tubercular disease, the other 2 dying of an acute lesion. Of the 6 who had relapse, 2 are now under active treatment with a prospect of a speedy cure, and 2 have been cured a second time. One has been in bed a year after excision of the joint, the other is on crutches. Of the 41 who recovered there is not a single individual who is incapacitated from doing a full day's work at his or her trade or occupation. Only one, a boy who had suffered from both Pott's disease and hip-joint disease, used a cane, and none used crutches. There are, among those who recovered, printers, glaziers, machinists, errand-boys, shop-girls, dressmakers, and many children attending the public schools—all at their work and none with evidences of active tubercular disease or any serious incapacity arising from the condition for which they were treated years before.

A CONSIDERATION OF THE NEUROMUSCULAR ELEMENTS IN HIP-JOINT DISEASE,

WITH ESPECIAL REFERENCE TO THE QUESTION OF THE ABOLITION OF PROTECTIVE TREATMENT[1]

A revision of a paper read before the Orthopædic Section of the New York Academy of Medicine, January 19, 1900 (see an abstract of the original paper and of the discussion in the New York Medical Journal for February 24th).

When a patient with hip-joint disease reaches a point in treatment where the question of convalescence is considered, one is met by conditions which are no less interesting or important than those which arise when a point of early differential diagnosis, involving the joint itself, is presented. Indeed, there is no condition in the treatment of hip-joint disease which calls for greater exercise of judgment, or which involves a closer estimate of that which is "organic," or due to the destructive osseous lesion itself, and that which is more purely "functional," or due to the treatment, than that which arises under these circumstances. As we must rely largely upon a neural sign in making an early diagnosis, so must we analyze the complex signs and symptoms which confront us in determining the ability of the oft-times damaged joint to perform its necessarily limited functions.

A tuberculous hip-joint—for it is the symptoms of chronic joint disease as found at this expressive articulation which I propose to consider—presents many peculiar features: The insidious onset of the disease; its oft-times uncertain history; the expressive inability of the patient, even in the first apparent stage of the disease, to bear the full weight of the body upon the affected limb; the frequent disappearance of the limp, in its early history, only to recur again, however, after some slight traumatism; the early appearance of the reflex muscular protection to the articulation; the reduced faradaic reaction of the muscles affected by this "spasm"; the concurrent appearance of the muscular atrophy; the existence of an increased muscular excitability; the frequent absence of pain; the changing character of the progressive deformities; the gradual disintegration of the bony tissues involved; the frequent destruction of the joint without abscess; the ordinarily innocuous character of the abscess when it does appear; and the long and persistent character of the disease, all point to the fact that we have in hip-joint disease and allied articular affections a complex pathological problem of much interest not only to the surgeon, but to the neurologist as well.

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[1] *The New York Medical Journal,* for April 14 and 21, 1900.

Indeed, throughout the entire course of hip-joint disease, especially, neural signs and symptoms of a suggestive type are present, which have not been adequately studied or satisfactorily explained. Orthopædic surgeons talk about "the reflex muscular spasm" as if it were an ordinary symptom. But it is not an ordinary symptom, and its nature has not been fully determined. Its value as a diagnostic sign is fully appreciated by the few whose studies have led them along certain lines of clinical work, but to the profession at large the expression "reflex muscular spasm" as applied to hip-joint disease, for example, conveys a very vague meaning, and is generally interpreted as a simple functional condition. Hence I feel perfectly justified in asking, What is the pathological interpretation of the marked neuro-muscular signs and symptoms which exist in hip-joint disease, and in allied articular diseases?

From a purely operative standpoint the problems involved in the treatment of hip-joint disease are neither difficult nor profound. They are well understood and are aptly taught in all our medical schools, and the mechanical aspect of its treatment, after many years, is beginning to receive the attention it deserves. The neuro-muscular elements are those which to-day demand a closer study and a more intelligent interpretation. The involuntary muscular joint protection, the early and expressive muscular atrophy,[1] the reduced faradaic reaction of these muscles,[2] and their increased muscular excitability,[3] are suggestive factors in hip-joint disease, and especially in those of the tuberculous variety. As long ago as 1872 I called attention to the reflex muscular spasm and the accompanying muscular atrophy, and later on demonstrated the reduced faradaic reaction of the affected muscles. I pointed out then (1876–'77) that these conditions must arise from a nerve irritation, and quoted from Brown-Séquard, Charcot, Paget, Weir Mitchell, and Seguin to sustain the position then assumed.

Recognizing that there was a specific muscular atrophy not due to disuse, and that the muscular protection was involuntary in joint disease, I sought in vain for a long time for an explanation of these phenomena, and it was not until I read Charcot's classical *Lectures on Diseases of*

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[1] Paget. *Clinical Lectures and Essays*, 1875; and Valtat, *De l'Atrophie musculaire consécutive aux maladies des articulations*, 1877.

[2] Reflex Muscular Contraction and Atrophy in Joint Disease, *Archives of Clinical Surgery*, June, 1877; also, The Ætiology and Pathology of Chronic Joint Disease, *Seguin's Series of American Clinical Lectures*, vol. III, No. 6, 1877 (by the author).

[3] E. G. Brackett. Reflexes in Hip Disease, *Boston Medical and Surgical Journal*, vol. cxxvi, No. 13, 1892.

the Nervous System that the matter became clear; and, in order that my present position may be made plain, I shall quote, as I did in 1877,[1] from the authorities which lea me to look for and to find a reduced faradaic reaction in the thigh muscles in hip-joint disease.

Charcot says, quoting from Brown-Séquard: "*Nerve irritation alone is capable of determining rapid and early atrophy of the muscles, preceded by decrease or disappearance of faradaic contractility. Complete nerve division does not induce atrophy and loss of electrical contractility until after an imcomparably greater length of time, as in the case of prolonged inaction.*"[2] (Italics are Charcot's.)

Charcot further says: "If lesions whose consequence is the abolition or suspension of the action of the nervous system are impotent to produce in distant parts other nutritive disturbances than those attributable to prolonged inaction, *it is not thus as regards lesions which determine, either in the nerves or nervous centres, an exaltation of their properties, an irritation, or an inflammation.*"[3] (Italics are Charcot's.)

And in a footnote this eminent author thus summarizes his conclusions: "It appears in fact to be demonstrated that the latter" (muscular alterations dependent upon nerve irritation) "supervene with much greater rapidity and are preceded and accompanied by more or less marked modification of electrical contractility, which do not show themselves in the former" (muscular alterations connected with functional inertia) "with the same characteristics and only make their appearance at the end of a very long lapse of time.[4]

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[1] *Op. cit.*

[2] *Lectures on Diseases of the Nervous System*, Sydenham edition, 1877.

[3] *Ibid.*

[4] *Ibid.*

Contributory to this subject also were the clinical observations of Paget.[1] Speaking of "the wasting of limbs near the affected joints," he says: "This wasting occurs quickly in nearly all acute inflammations of joints. More slowly in chronic inflammations. . . . It is not, I repeat, a mere wasting from disuse. . . . I wish I could explain it better than by calling it reflex atrophy."

And Weir Mitchell, whose contributions to this subject of irritative nerve lesions is so well known, wrote me, November 5, 1877, as follows: "There is a resemblance between the two conditions you speak of" (the muscular spasm and atrophy occurring in joint disease and the same conditions following injuries of nerves): "both sets of contractures and atrophies result from irritative peripheral lesions of nerves in and out of joints."

The late Dr. E. C. Seguin, who kindly aided me on many occasions in my investigations, and who publicly demonstrated my experiments, took the same ground regarding them as did Weir Mitchell.

This was done without any knowledge on my part of Valtat's[2] experimental work. Indeed, our contributions occurred simultaneously, Valtat's in France, my own in this country, and our work, which might to some appear to be similar, varied in this important respect: Valtat demonstrated that an acute joint inflammation artificially produced in animals, was followed by the muscular atrophy so well described clinically by Paget, while the writer, having long recognized this atrophy as of constant occurrence in especially the chronic form of articular disease, demonstrated the reduced faradaic reaction of the muscles in these conditions, and pointed out in cases of psuedo hip disease, where the patients had been subjected to a prolonged immobilization treatment, that the reduced faradaic reaction was absent, and that the atrophy was simply that of disuse.

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[1] *Clinical Lectures and Essays*, D. Appleton and Company, 1875, pp. 208, 209.

[2] *Op. cit.*

On various occasions[1] after the essays of 1877 I have called attention to these matters. I venture to do so again, and on this occasion I shall attempt to describe the condition which we call "reflex muscular spasm," more in the hope, however, of eliciting a discussion than of being accurate in my description.

The peculiar muscular condition which is manifest, especially in the first stage of hip-joint disease, and which persists more or less throughout the entire course of the disease, is characterized by a sudden, almost peremptory, contraction of the muscles involved when passive motion is attempted beyond a certain point. A peculiarity of this contraction is that the muscle or muscles are quiescent until a certain point is reached, when this instinctive muscular resistance occurs. This resistance (muscular spasm) is reflex and involuntary, being wholly beyond the control of the patient. It is not modified by large doses of opium or chloral, and it is only annulled by the anæsthesia of ether or chloroform. It is progressive, increasing in untreated cases until, sometimes, ankylosis is simulated. It produces the various deformities of the articulation and is accompanied by a specific muscular atrophy, not due to simple inertia. This "reflex muscular spasm," as well as the muscular atrophy, is a distinct pathological expression of the disease. Together they present the essential phenomena which accompany nerve irritation, and the conditions which characterize the spasm place it under the head of a *tetanoid* spasm; a localized tetanus, so to speak, which is excited by intra-articular pressure, just as the same muscle is subject to the faradaic tetanus of Landois when influenced by the interrupted current.

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[1] *Pott's Disease: Its Pathology and Mechanical Treatment*, G. P. Putnam's Sons, 1879; *The Hysterical Element in Orthopædic Surgery*, G. P. Putnam's Sons, 1880; Lecture on the Pathology, Symptoms, and Diagnosis of Ankle-Joint Disease, *Annals of Anatomy and Surgery*, January, 1882; On the Principles of the Mechanical Treatment of Hip-Joint Disease, *New York Medical Journal*, November 23, 1889; What are the Indications for the Discontinuance of the Mechanical Treatment of Hip-Joint Disease? *New York Medical Journal*, November 28, 1891.

As the spasm of true tetanus is modified or annulled by chloroform, so is the spasm of the muscle in hip-joint disease; and as the spasm of tetanus returns after the effects of the chloroform have passed away, so it does in hip-joint disease; the reflex excitability of the muscle in hip-joint disease is increased, so it is in tetanus. In all important respects the analogy between the muscular conditions in tetanus and in hip-joint disease holds good. This spasm is tonic, it is persistent and unyielding. It does not vary under the conditions noted except that, if untreated, it becomes more and more pronounced as the osseous structures become more involved.

A brief definition of this reflex muscular condition may therefore be stated as follows:

An involuntary, tonic muscular contraction, tetanoid in character, having its origin in nerve irritation caused by intra-articular pressure. It is characterized by a specific muscular atrophy and a reduced faradaic reaction of the muscles affected. It is accompanied by a peculiarly persistent and resistive element, which yields only to ether or chloroform, and it is emphasized by an increased muscular excitability.

In a lecture on The Pathology, Symptoms, and Diagnosis of Ankle-joint Disease,[1] delivered at the New York Orthopædic Dispensary and Hospital on November 3,

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[1] *Op. cit.*

1881, I used the following language: "If you flex and extend an inflamed ankle-joint with osteitis, you will find in many cases a very considerable degree of movement. But before the normal limit is reached the muscles which oppose become suddenly *tetanized.*" I have never had occasion to question the accuracy of this statement of nineteen years ago.

And we need not look far for an explanation of these symptoms. It is generally conceded that hip-joint disease is an osteitis, that it has its origin in the epiphysis, and if we stop to consider that this epiphysis is a local centre of growth and development, supplied with nerve filaments from the three great nerve centres—the cerebral, the spinal, and the great sympathetic—as has been demonstrated by several eminent authors, Kölliker, Luschka, and Kobelt among them, we can readily see that the presence of a foreign body like the bacillus of tuberculosis would alone be capable of creating a nerve irritation, and that passive movement beyond a certain point would produce an intra-articular pressure which Nature would resent. This nerve irritation would also, I think, account for the muscular atrophy, for the reduced faradaic reaction, for the tetanoid character of the muscular spasm, and for the increased patellar tendon reflex—all of which are present, as a rule, both as early and late symptoms, especially of the tuberculous degeneration of the epiphysis. It is often difficult, however, to demonstrate the patellar tendon reflex in patients when the immobolizing treatment has kept the knee extended for a considerable length of time.

In whatever light this reflex spasm is regarded, one fact must be remembered. The universally acknowledged principle of immobilization in the treatment of hip-joint disease brings an additional element of confusion into the resulting conditions, for it can not do else than add to the muscular atrophy and disability of the limb, and make still more difficult the question as to when the joint is in a condition to resume its ordinary functional activity. To the recognized effects of the disease itself we must add that of prolonged disuse, bandaging, etc. The mechanical treatment produces an interference with the normal physiological processes of circulation and growth. A weakened member is thus added to the diseased and damaged joint and unnecessarily, I think, confuses us when we come to consider the problem of re-establishing, so far as possible, the normal relation of the affected limb and joint to the rest of the body.

I know of no better way of bringing out the points I wish to illustrate than to refer to a condition which is frequently seen at the knee-joint.

We have all been called upon to decide whether a knee-joint, having been incased in the ever-ready gypsum splint, after a sprain or wrench, with or without synovial effusion, was in a condition of serious, perhaps tuberculous, chronic inflammation. Whatever the original condition was, the clinical picture presents a sensitive joint with altered outlines, accompanied perhaps by pain and heat, but always by stiffness. The articulation is not in a condition to perform its normal functions, and the disability is such as to make normal locomotion impossible. The muscular symptoms resemble the reflex muscular spasm of a tuberculous joint; muscular atrophy is present, and the condition suggests a chronic inflammation of a serious type.

Oft-times this condition is the result of a too prolonged immobilization of the joint after some trivial injury. As soon as the joint is released from its enforced imprisonment and is given a chance to resume its normal use, recovery takes place.

While in these patients, as a rule, no tuberculous disease is present, the immobilization treatment, added to a slight traumatic lesion, produces a condition which may closely resemble it; and I have seen patients in this condition where it was very difficult, after months, and sometimes years, of immobilizing treatment, to make a positive diagnosis at one examination. But once the condition has been recognized the treatment is plainly marked out. Use, exercise, massage, etc., soon re-establish the lost functions of the joint.

In a typical case of hip-joint disease in childhood or adolescence we are, generally speaking, dealing with an infectious osteitis involving an epiphysis. If to this clearly recognized pathological condition we add the effects of years of immobilization, etc., the disturbed nutrition and growth, due to the treatment alone, become interesting and even disturbing factors in the general result. While we can not ignore the presence or the influence of this element, I am prepared to say that the one who regards it as important, when compared with the result of the disease itself, makes a serious error; just as one who fails to recognize the presence of a latent but really existing tuberculous disease will be sure to bring about a relapse by prematurely giving the joint too much liberty.

The knee-joint above referred to is weak, the products of an acute non-infectious process are present, and it needs active, not immobilizing, treatment. When a diseased hip-joint reaches the point of true convalescence, this same treatment is required, and recovery rapidly takes place. But if tuberculous disease in an insidious form is present, the joint requires protection just as much as when the more pronounced symptoms are evident. And it is during this stage of apparent convalescence that many errors have been made.

It is during this stage of apparent convalescence that the claims of the patient and his friends assert themselves. They demand for the joint use and exercise. The ordinary lay mind, and sometimes the medical mind, sees only the wasted limb, the shortened member, the weak articulation, and the disability. The long absence of apparently important symptoms, the prolonged treatment, the continued and enforced disuse of the affected joint and leg have but one meaning to them, and they see cause and effect from the standpoint of the simple sprained ankle or the non-infectious, traumatic inflammation of some of the minor articulations. The question, however, which we have to meet is: Is the joint really well, but weak and vulnerable, or is the disease latent, ready to reappear if the joint is removed from the influence of local mechanical protection?

Between these two conditions the surgeon must decide. To condemn a simple sprained or overtreated joint to further immobilizing treatment is clearly doing an injustice to the patient; just as it is wrong to expose a patient in the quiescent but uncured stage of a progressive tuberculous osteitis to the certainty of a relapse. The former condition, confusing as it is at times to the most acute observer, has added undeserved fame to the Christian Scientist, the bone setter, and the osteopath; while the latter has brought undeserved criticism to some of us who have been only too glad to hope, in a questionable case, that the experimental removal of protection to the joint would be followed by good results.

The contrast between the simple traumatic knee and the infected epiphysis at the hip is one that must strike the most casual observer. On the one hand, we have an uncomplicated, non-infectious arthritis, which in its muscular signs and symptoms resembles the joint affected with tuberculous disease. But pathologically there is this great difference between them: In the simple traumatic arthritis—osteitis, if you like—there is no destruction of cancellous tissue and the muscular condition improves with use after the more acute symptoms have subsided; while in the tuberculous epiphysitis there is a distinct involvement of the cancellous structures by an infectious germ, which only needs the element of ordinary use to further develop and multiply the bacilli, and thus increase the area of destructive invasion.

As I have already remarked, the epiphysis is a local centre of growth and development, supplied with nerves from the three great systems. The development *within* the epiphysis of a focus of tuberculous disease, especially at one of the centres of ossification, presents a far different pathological problem from that which occurs as a result of a simple injury acting from *without* the joint with no germ present.

The nerve irritation, and hence the tetanoid spasm, is more pronounced, more persistent, and far more expressive in the former than in the latter. So also we find that the chronic tuberculous degeneration of the synovial membrane does not produce either a reflex spasm or the rapid muscular atrophy of a tuberculous osteitis. The synovial membrane has comparatively few nerves, and, being a simple serous tissue, is not a local nutritive centre, and is further not subjected to intra-articular pressure during passive movements of the joint.

The more I have studied the neuro-muscular signs and symptoms of chronic joint disease, both from a clinical and a pathologidal standpoint, the more I am impressed with the correctness of the position I took in 1877, and the more certainly does the existence of this tetanoid spasm indicate that the affected joint is something more than simply weak and vulnerable. One thing has certainly been demonstrated (and this could not be said a few years ago), and that is, that the long-continued absence of pain, the relief or correction of the deformity, and the existence of a considerable improvement in the passive motions of the joint do not necessarily indicate that the stage of true convalescence has been reached.

Undoubtedly these conditions are Nature's indication of an improvement in the pathological condition of the joint and that a slow but sure repair is going on. The tuberculous element is modified, perhaps essentially modified, but we can not always build our hopes of complete repair upon this evidence, especially when the bony structures have been, as they almost always are, altered by disease.

That these conditions are not fully appreciated by the medical profession at large is evident from some recent experiences of mine, and it will not be amiss to relate a couple of them.

Three years ago I saw in consultation with Dr. J. William White, of Philadelphia, a little girl, five years old, who had well-marked hip-joint disease with deformity and abscess. After a very carefully conducted course of treatment, with the use of the traction splint, under Dr. White's supervision the joint condition had improved to such an extent that Dr. White kindly asked my opinion as to the propriety of discontinuing the protective apparatus then in use. Upon examination, I found no deformity; good motion of the joint in all directions; flexion to ninety degrees; rotation in and out about half the normal; abduction and adduction limited, but very distinctly present, and extension of the thigh stopped in the long axis of the body. The abscess had healed and there was an inch of shortening. The patient's general health was excellent. I gave my opinion that the condition of the joint did not warrant, even with these favorable symptoms, any modification of the protective traction apparatus. My reasons for giving this opinion were based upon the following observations: First, the inability of the patient to bear her weight upon the affected joint without the "flinching" so characteristic of the first stage of the disease; and, secondly, the marked presence of the tetanoid spasm when the attempt was made to exceed the limit placed by this instinctive muscular protection, in the various passive movements of the articulation. Perhaps this spasm was less marked than it is when seen in the first stage of the disease. But the difference was in the quantity rather than the quality, and it was so uniformly present, and so consistently aggressive when subjected to the ordinary diagnostic test, that no one accustomed to make these tests could hesitate. So also the expressive disability of the joint pointed to more than the functional derangement produced by mere disuse. The condition of the joint seemed very favorable to a most satisfactory recovery in time, but it was plain that the real stage of convalescence was not present. I advised the further use of the protective apparatus, an opinion which Dr. White also entertained, and we instructed the patient to continue the apparatus and to use crutches as an additional protection.

It became necessary for the patient to accompany her mother to the Pacific coast last winter. After being there for some weeks a slight accident to the patient led the mother to consult a local surgeon of excellent repute. Apparently disregarding the symptoms which were so evident and so suggestive, he declared the patient to be well, and advised the removal of the apparatus and a practically unlimited use of the leg and joint. The patient, only too willingly perhaps, followed his advice. When she returned in the spring I examined her again. The hip joint was practically rigid, the thigh was flexed and adducted, the shortening had increased three eighths of an inch, the tetanoid spasm was very marked, and pain was present.

Another history presents some interesting features. Four years ago a girl of fourteen years was brought to me with hip-joint disease of two years' duration. The onset of the disease had been so insidious that several surgeons had been deceived, especially by the intermittent limp.

Several times during these two years the limp had entirely disappeared, and, the other symptoms being deemed unimportant, no special diagnosis was made. It was not until a sudden avalanche of symptoms occurred, the outbreak which so often marks an insidious invasion of this nature, that the real condition was recognized.

When the patient came under my care the affected limb was an inch and three quarters shorter than its mate; marked deformity, atrophy, and persistent tetanoid spasm, practically limiting all motion of the joint, were present. A careful mother and an intelligently supervised home treatment brought the patient last summer to a point in recovery where the outlook for a most excellent result was very promising. There were about forty-five degrees of motion in flexion and extension of the thigh. There was slight adduction, with flexion of the thigh. The shortening had not increased under treatment. A test of walking at that time without the splint was made, and the expressive flinching of the diseased joint was very evident. The question of the removal of the splint was discussed at that time, and I gave my opinion that the joint was not in a fit condition to bear the weight of the body.

Under these circumstances the patient went abroad, where it would seem she continued to do well, with a protective perineal splint without traction. Hearing much in London of an eminent surgeon, the mother availed herself of the opportunity and consulted him. He advised the mother to discard the apparatus and to give the child unlimited use of the limb and joint. He even doubted that hip disease had ever really existed. His advice was positive and unqualified, and naturally impressed the mother very much. But she declined to take such a radical step without further conference with me. Upon the patient's return I examined her carefully. All deformity had been removed except that the thigh still remained somewhat adducted and slightly flexed. Her gait had slightly improved also. There was a modification of the "flinching" while walking, and the shortening remained the same. But there was in every movement of the joint, as soon as the limit was reached, a distinct tetanoid resistance, and any effort to exceed this limit produced a distinct muscular spasm, though there was no oral expression of pain. In short, the symptoms of the first stage of the disease were present, showing that there had been a most satisfactory improvement and a rapid stride toward recovery.

The mother frankly told me of her experience with the London surgeon, but I was unable to coincide with him and advised the further use of the protective apparatus. My advice was accepted and the patient continues to improve, both as to the amount and quality of joint movement.

I have no doubt whatever if the advice of the London surgeon had been followed that the result which occurred in the patient referred to above would have taken place in this instance.

It is not my intention to tire you with a repetition of similar cases in detail. Some of them, however, have been very instructive. I have seen abscess and death follow the result of a formal consultation under these circumstances, the removal of the protective apparatus being practically insisted upon by the father of the patient and the family physician. I have been obliged in some instances (the patient being removed from my care) to sit calmly by and witness the downfall of years of patient work, and I have myself taken risks, and have on some occasions permitted, rather than advised, the discontinuance of the protective treatment at a critical moment, when subsequent events proved my error in so doing. My best results have been in those cases where I have insisted on the continuance of the protective treatment for a long period, or until I was satisfied that the true reflex muscular spasm had ceased to exist, and until I felt that the damaged joint was in a stage of true convalescence.

I have never seen anything but good result from this prolonged treatment. A shortened limb without deformity and with considerable motion is not a calamity, and even if there is ankylosis in a good position the result is a satisfactory one. A limb which is badly flexed and adducted, even if there is no bony shortening, is a vicious deformity which is not even relieved by osteotomy unless the joint is sound and capable of bearing the weight of the body. I have seen cases of recurrent flexion and adduction of the thigh after osteotomy. Our aim should be to cure the joint disease, and not simply to relieve one of its signs, and if one can maintain the protective treatment for a sufficient length of time one will secure the best attainable result, which in the great majority of cases is a very useful member with motion in many cases, and always without any marked deformity.

On many occasions in my lectures and elsewhere I have called attention to the expressive character of this tetanoid spasm in joint disease. It forms, I think, the only reliable sign both in diagnosis and treatment. It enables us on many occasions to make a prognosis. Its presence also enables us to detect the disease in its first apparent stage, and the modification or persistence of this peculiar telltale neural sign is our principal guide in treatment. And I feel perfectly justified in saying that we can not afford to ignore the presence of this tetanoid spasm at the diseased joint, either in the first or last stage of the disease. We must, however, be prepared to recognize the difference between the true tetanoid spasm and those conditions which simulate it. A joint may be sensitive and the muscles rebel against motion, but this is not the tetanoid spasm of tuberculous joint disease. A joint may be "hysterical," or the subject of a traumatic neurosis, but the muscular conditions are, as a rule, a poor counterfeit of the genuine. A joint may be vulnerable and its muscles may resist passive movement, but it is not the involuntary muscular resistance of a chronic articular osteitis. A joint may be irritated, even inflamed, by traumatism, but the instinctive quality of the true tetanoid spasm is absent.

It is granted that the distinction between these conditions, especially if the history is obscure, is difficult under certain conditions. But these are mostly questions of diagnosis before treatment has been inaugurated. The subject under consideration merely raises the question as to whether a well-recognized condition of reflex muscular spasm which was clearly present in the early stages of the disease is still present. If it is present, what does it signify? The only other question is the one that involves the muscular state which follows long disuse, and this I am sure is not a difficult problem, for prolonged disuse, such, for instance, as is seen in the knee-joint after its continuous immobilization with the hip during treatment, does not suggest reflex muscular spasm. We are apt to have a lax knee-joint rather than a rigid one under these circumstances, and I have never known any one to mistake the easily overcome muscular resistance at the knee, when it occurs under these circumstances, for the muscular conditions accompanying disease of the hip.

When, then, does convalescence really begin in hip-joint disease? Certainly not so long as the most important symptom which enables us to make a diagnosis is present; certainly not so long as the joint disability is so pronounced as to make the act of locomotion a greater effort than that occasioned by simple disuse; certainly not when deformity is present, and certainly not when the demand comes from an unnecessary fear on the part of the patient or his friends that the prolongation of the protection to the joint is going to produce a useless limb.

It is not the function of this brief essay to deal with the question of the especial form of the protective apparatus to be used after the more urgent symptoms and signs of the disease have been mitigated by treatment. But there is one point which I wish to emphasize. One great danger to the joint and limb lies in a too prolonged immobilization. In other words, an actual immobilization is not necessary after a certain amount of motion has been reached as the result of this immobilization. On another occasion[1] I submitted the following proposition: "Within the limit set by Nature (*i.e.*, by the reflex muscular spasm), motion I believe to be harmless."[2] Call this a dictum, if you please, but it is the dictum of experience, and I wish to emphasize it on this occasion. And I may say as an additional dictum based upon the same experience, and amplified by ten years of further observation, that it is our duty to adapt our protective apparatus to the changed conditions brought about by treatment. The only novelty in this proposition lies in limiting the motion of the apparatus *inside* of the point fixed by the tetanoid spasm. This can be done, however, very easily by any one, and if this rule is followed we shall have much better results, much sooner obtained results, better joint motion, and a more useful limb, without deformity and with less bony shortening. But the surgeon who carries out a strictly-speaking immobilization treatment to the end will find the problems raised in this paper to be more difficult of solution in every way, and he will imperil the result he is most anxious to obtain—namely, a joint without deformity and a fair amount of motion.

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[1] On the Principles of the Mechanical Treatment of Hip-Joint Disease. *New York Medical Journal*, November 23, 1889.

[2] This statement, to a certain extent, has been emphasized by Dr. Brackett, who says (Reflexes in Hip Disease): "The table in this series of cases shows an almost constant relation between the spasm and the increased condition of the reflexes, and granting a value to one we must give the same to the other. The indications are that it is an evidence of an irritable condition of the joint rather than a sensitive condition, and would apparently be of value in determining the amount of freedom to be allowed the diseased joint."

But to remove all apparatus and to place the patient on his own resources during the apparent, not the real, stage of convalescence is a serious error. To condemn a patient to a uselessly prolonged mechanical treatment is a confession of ignorance, but it is a greater confession of ignorance to remove a necessary protective apparatus when the symptoms indicate its further use. And further observation and experience enable me to emphasize the statement I made in 1891[1] before this body—namely, "I believe that in all but the exceptional cases, a relapse as to the deformity or the disease, or both, is likely to occur as the result of the traumatism of ordinary locomotion, unless the proper mechanical protection is maintained until the articulation is free from true reflex muscular spasms or is ankylosed."

When this point is reached and the joint is free from reflex muscular spasm, the problem is no more difficult of solution than that which presents, for instance, in the traumatic, non-infectious, knee inflammation already referred to. The joint may be placed at once upon its own resources with a gradually increasing use of the now simply weakened limb, and by the judicious use of electricity, massage, etc., the limb and joint will soon recover from the effects of the prolonged disuse. The muscular atrophy may never wholly disappear, but the limb as a whole will do its work wonderfully well, and if, as we have a right to expect in many cases, a considerable motion returns to the joint as a result of our treatment, the patient will walk without a limp, and in those cases where ankylosis occurs without deformity a most useful limb will result.

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[1] *Op. cit.*, *New York Medical Journal*, November 28, 1891.

ON THE REMITTENT LIMP IN THE FIRST APPARENT STAGE OF HIP-JOINT DISEASE, WITH REMARKS ON THE EARLY DIAGNOSIS OF THE DISEASE[1]

Read before the American Orthopædic Association, Philadelphia, June 5, 1902.

On the 12th of November, 1901, I saw in consultation with Dr. J. F. Lyon, a boy, J. V., aged six, who presented the following history:

Early in April, 1901, the patient, after a slight accident, commenced to limp. The limp was quite pronounced, but was unaccompanied by any pain. The patient was a very active little fellow—a sort of leader among the children of his neighborhood, a quick runner and a good jumper. His parents thought at first that the limp was occasioned by the slight sprain referred to. But little attention was paid to the matter for the first few days, but as the limp persisted and was worse after rest, and especially so after getting out of bed in the morning, it was thought that the condition might be due to rheumatism, and this view was emphasized by the fact that the limp was favorably modified by exercise. The boy essentially, and I think instinctively, modified his activity during this time and voluntarily gave up his most active out-door exercises. After the first ten days, the limp steadily improved and finally disappeared in about two weeks. He then gradually resumed his wonted excessive activity and again became the leader of the street in "stunts" of various sorts. The parents and neighbors assert positively that the limp entirely disappeared and that the boy became as agile and as active as before the attack. No treatment whatever was given.

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[1] *The New York Medical Journal*, for August 16, 1902.

Nothing unusual presented in the history of the boy until the August following, when he again commenced to limp. On this occasion there was no apparent cause—that is, the limp could not be traced to any distinct sprain or wrench. It was supposed to be rheumatism by the parents, who, as on the previous occasion, did not seek professional advice. The child had no treatment whatever. The limp was pronounced, more so than in the previous attack, and presented, generally speaking, the characteristics mentioned above. It was also a little more persistent, and nearly a month went by before recovery occurred. The incident did not attract special attention. The boy had simply, in the parents' minds, recovered from a slight attack of rheumatism. So far as the parents could see, recovery was complete and the boy went on the street again to his usual sports and games.

About a month before I saw the patient, he had had a third attack of limping, which was again disregarded by the parents. The matter attracted only casual notice, as he had already recovered from two previous attacks without professional advice. On this occasion, however, the symptoms were more persistent; the limp became gradually worse, instead of gradually better. After a while the patient could scarcely walk, and yet he had no pain. Locomotion became very awkward, then difficult, and finally an explosion of acute symptoms occurred. When I saw him he was in extreme pain, the thigh was flexed badly, abducted, and rotated outward, and any movement of the bed even was followed by excruciating pain. In short, the patient was suffering from hip-joint disease in its most acute stage.

I venture to cite this interesting history because it is typical of a large number of cases which have come under my observation, and because I think that this early remittent limp is not as yet sufficiently recognized, nor is its importance fully appreciated by the medical profession, although Barwell in his classical work[1] says: "The limp therefore may be absent a week or so and then return, perhaps to remain permanent, perhaps again to intermit."

On the occasion of the third annual session of this association, held in Boston in September, 1889, I read a paper "On the Principles of the Mechanical Treatment of Hip-Joint Disease"[2] in which I say: "It is well known that in this first stage of hip-joint disease—the stage of vulnerability, . . . the limp referred to as occurring in the first stage may disappear . . . I have known this to occur seven times in one patient before the symptoms become so pronounced that a diagnosis was made—the surgeon in attendance being unfamiliar with the fact that a remission of the limp was likely to occur in the early stage."

It would seem that I was mistaken in saying that the remittent limp was "well known." One of my hearers—a prominent member of the association, in the discussion which followed, said: "The statement has been made to-day that in certain cases the limp disappeared. . . . I confess I have never seen a patient who once began limping with hip disease lose the limp until the case had been some time cured."[3]

The only other reference to the matter at the Boston meeting in the prolonged and interesting discussion which followed a Symposium on Hip-Joint Disease (my paper being one) was made by Dr. Herbert L. Burrell, of Boston, who said: "There is one point I should like to refer to, and that is with reference to the subsidence of the limp during the course of the disease. . . . I feel quite sure I have seen this in certain cases, not, however, without treatment. We have cases that come to the Children's Hospital where it is difficult to make a diagnosis. The child perhaps has a slight limp; we send it home and request that it be kept in bed a week. The mother returns with the child, there is no limp, and the limb remains this way for possibly a month or two, when the child returns with a recurrence of the limp."[1]

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[1] *A Treatise on Diseases of the Joints.* By Richard Barwell, F.R.C.S. 2nd Edition, London, Macmillan & Co., 1881, p. 437.

[2] *Transactions of the American Orthopædic Association*, Vol. II, 1889; also *New York Medical Journal*, November 23, 1889.

[3] *Ibid.*, page 191.

I was led to say in my paper that the remittent limp was well known because Barwell and others had described it, and I had taught that it was a frequent factor in the histories of many patients coming under my observation, and I have been in the habit of emphasizing the value, as well as the danger, of this significant manifestation of the first apparent stage of hip-joint disease, both in my lectures at the New York Orthopædic Dispensary and Hospital (1876–1898) and in my lectures at the University Medical College (1882–1886). These facts are my excuse for bringing the matter thus prominently to your attention.

More recently, however, the matter has received attention. Bradford and Lovett, under a special caption, "Remissions," in their chapter on Hip Disease, say: "Any account of the symptoms of hip disease would be incomplete without speaking of the remissions in the course of the affection. In the early stage this is especially noticeable, and a patient may, to outside appearances, entirely recover from the symptoms of pain, lameness, and discomfort for some days or weeks."[2]

Whitman says: "In the early stage of the disease the limp may even be intermittent, although it is probable in most instances some change from the normal gait may be detected by the practised eye."[1] Quite a number of works on orthopædic surgery do not mention the remittent limp.

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[1] *Op. cit.*, page 193.

[2] *A Treatise on Orthopædic Surgery.* By Bradford and Lovett, 2nd Edition, New York, William Wood & Co., 1899, p. 222.

These quotations are, however, sufficient to show that the remittent limp is not, as a prominent general surgeon once said to me, "a creature of your own imagination." On the contrary, it is a most important sign of the early stage of the disease, which, taken in connection with other signs and symptoms, enables one to establish an early diagnosis and in a certain number of instances to abort, or at least to shorten, the course of the disease.

In the paper already referred to I also state as follows: "It has happened several times in my experience that the limp has disappeared without actual treatment while the patient was under observation."[2] This distinctly emphasizes Dr. Burrell's statement, already quoted. I further remark: "I have watched these cases with much interest and I have found that *the muscular protection of the joint did not wholly disappear as the limp did.*"[3] Indeed, in this essay I dwell at some length upon especially the diagnostic value of the reflex muscular spasm in tuberculous disease of the hip in all its stages. Some of the important views there expressed have been pretty generally accepted, if not adopted, by some of the recent writers on orthopædic surgery.

The important point, however, to be established is that a limp, whether accompanied by pain or not, in childhood, even if it has an apparently direct traumatic cause, should not be passed over lightly. It would be well if all parents knew that a slight limp may mean the first apparent stage of hip-joint disease, even if it should wholly disappear. I take occasion, both in my college lectures and in my lectures to the trained nurses at St. Luke's Hospital, to point out this fact, and I trust every member of this association will make use of suitable opportunities to make the fact more generally known.

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[1] *A Treatise on Orthopædic Surgery.* By Royal Whitman, M.D., Philadelphia, Lea Brothers & Co., 1901, p. 227.

[2] *Op. cit.*

[3] *Op. cit.*

I venture, in this connection, to recite a bit of history. During the summer of 1872, while in my first provisional year of service as the orthopædic surgeon to St. Luke's Hospital, a patient, a girl, four years old (L. G.), giving the history of a remittent limp, with slight muscular resistance in all directions, was under the observation of the late Dr. James L. Little and myself in the children's ward of the hospital. The symptoms were so slight and the disability was so trivial that Dr. Little questioned my diagnosis of hip-joint disease. As a matter of test, and at my suggestion, we administered ether and the muscular protection of the joint disappeared, only to return, however, before the patient was wholly conscious, and before we left the operating room Dr. Little asked me to explain this muscular phenomenon. I said: "The muscular symptom, as shown by the ether test, is a purely reflex affair, and the muscles are affected with a true reflex spasm." So far as I know, this is the first occasion where the term "reflex spasm" was applied to the conditions existing in hip-joint disease. I certainly had never heard it used by anyone, and thenceforth I used the term "reflex spasm" in describing the conditions now so familiarly known by us all. I know that Barwell with his acute reasoning mentions "neuromuscular irritation and contraction" as being the factors which "fix the thigh at certain lateral and antero-posterior angles with the pelvis."[1] And he also refers to the "spasm of certain muscles" in connection with "the dull aching and with the sharp intermittent pains"—in other words to the "spasms" which are "chiefly prevalent at night when the patient is sinking to sleep,"[1] but nowhere in his writings have I found any reference to the "reflex muscular spasm" as we understand it to-day.

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[1] *Op. cit.*, page 430.

I believe that the first real demonstration of the true nature of the reflex character of the muscular condition at the hip, in hip-joint disease, occurred in St. Luke's Hospital, under the condition named, in 1872.

While I feel privileged to make this statement, I shall always be glad to waive any claim to originality or priority.

[1] Barwell, 1st Edition, 1861, page 308.

A CASE OF HIP-JOINT DISEASE, COMPLICATED BY A FRACTURE OF THE FEMUR; ADAM'S SUBCUTANEOUS SECTION OF THE NECK OF THE FEMUR; DEATH.[1]

THE following case presents some very interesting and peculiar features, both in a mechanical and surgical aspect. From the very lengthy history of the patient, as it appears upon the records of the New York Orthopædic Dispensary and Hospital and of St. Luke's Hospital, New York, I gather the following facts, which comprise the most essential points:

Sarah S., aged five years, a resident of New York City, was received as an out-patient at the New York Orthopædic Dispensary and Hospital on June 20, 1870, coming under my personal care. Her father, an English seaman, and her mother, also English, were apparently healthy people. The former had suffered occasionally from rheumatism, but there was no trace, so far as could be ascertained, of any hereditary taint. The brother and sister of the patient were very strong and healthy, and the patient herself showed no especial evidence of struma, though she was paler and less robust than the other children.

When the patient applied for treatment, the mother stated that the cause was supposed to be "a violent fall upon the sidewalk." The examination of the patient developed the fact that she was suffering from hip disease, the left hip being affected. The history shows that she had been suffering for two years prior to her admission. The thigh was flexed and adducted, and the patient was walking upon crutches. There was very limited motion at the hip-joint, movement being arrested at a certain point by a tetanic spasm of the muscles, whenever the limit of motion was reached. There was no abscess visible, and none could be detected by the most searching palpation.

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[1] *Annals of Anatomy and Surgery*, December, 1883.

The patient had been treated in a public institution of this city, where several blisters had been applied and a spica bandage had been worn. The effect of this treatment was a modification of the pain and an increase of the deformity.

A few days after the history had been recorded, I applied a long extension splint, at the residence of the patient, placing the limb upon an inclined plane, and using an abduction screw. On August 19, 1870, about two months after the patient applied for treatment, "the flexion and adduction were nearly overcome." On August 29, "the improvement is remarkable." On September 23, "excellent motion." On October 4, the patient was permitted to "run about out of doors," using the apparatus as a protection. This privilege was much abused by the patient and her mother. On November 17, I find the record, "does not receive proper care; large, hard tumor in groin; no fluctuation." December 9, "has symptoms of abscess." January 4, 1871, "has dermatitis from the adhesive plaster; name selected for the corps of lady visitors." February 15, "has better care since the ladies visited patient; is improving." March 6, "œdema still exists at hip." April 17, "œdema still at hip; no pain; good motion." May 2, "motion better; symptoms of abscess disappearing." July 10, "joint motion quite free."

This history illustrates a not unusual course of a patient in dispensary orthopædic practice. Feeling the great relief which the apparatus affords, and the deformity being overcome, these patients do too much. They become as active as other children who are not afflicted with joint disease. They are liable, sooner or later, from this cause, viz.: over-exertion, to have a relapse. They build up again by rest and care, as this patient did, to a point where with care and attention a very excellent result may, with reason, be expected. But this patient was so extremely active, that, notwithstanding the cautions and rebukes administered to the mother, the patient had her own way, with, in this case, a very unusual result.

July 23, 1871.—"Patient had a fall and fractured the femur, on the diseased side, at junction of lower and middle-third. The apparatus was uninjured." *Treatment.* —Removal of adhesive plaster on thigh to a point just below fracture. Traction with the hip splint, the fractured ends being held in apposition by a leather splint, which was snugly bandaged to the entire thigh. Patient was seen every day, and good traction was maintained. On August 25, it was found that the fracture had united perfectly in good position, and that there was one half inch shortening, some, if not the greater portion, of which was due to the disease.

The patient was now put upon her feet, and she again commenced to walk about, using the apparatus, of course. On November 17, "the motion at joint good; no symptoms of abscess present." Several unimportant entries show that the patient continued to improve up to December 19, 1873, when the apparatus was removed and the patient was kept under observation to test the effect which would follow the removal of the support. On January 19, 1874, it was deemed advisable, on account of slight pain and reflex spasm to again apply the apparatus.

On April 14, 1874, the apparatus was again removed, and after watching the patient closely until September 10, 1874, she was discharged cured, with almost perfect motion at the joint, one and one quarter inch shortening and a very slight limp. On November 18, 1875, the patient was visited. There had been no relapse; "general condition good; considerable motion at joint; one and one quarter inch shortening."

This period of the patient's history is very instructive.

After reaching a point where recovery was almost certain, she fell, and, by some strange combination of circumstances, broke the thigh bone that was protected by a strong extension splint. I anticipated much trouble in this case, and looked for a decided increase of symptoms. But by making a considerable amount of traction and using a coaptation splint, I succeeded in obtaining a good result as to the fracture, and protected the joint at the same time. There was no decided increase in the symptoms, and, as shown in the history, after a somewhat prolonged struggle, the patient was discharged cured, with good motion, and with an excellent and useful limb.

This experience with the hip splint in the treatment of a fractured thigh, and the favorable result obtained, under so many discouraging circumstances, led me soon after to adopt the same measures in a case of ununited fracture of the thigh, in an adult, aged 55, who had four months previously been thrown from his carriage. The surgeon who attended the case immediately after the fracture used the gypsum bandage.

When I saw him there was three and one half inches shortening, and the point of fracture at the junction of upper and middle thirds was easily demonstrated. I applied a long extension splint, and drew the limb down to nearly its normal length, and maintained it there for four weeks. Then a modified support was applied, and in four months union was complete, with about one inch shortening. Another case very similar to it in all essential particulars came under my care two years ago, and was treated in the same way. The result was perfect union in three months. A case of recent fracture of the thigh was also treated by Dr. George A. Peters, in St. Luke's Hospital by this method, in a child, with an excellent result.

As to the course pursued by the patient after the last record made above, we have no positive evidence. It is to be presumed that the child followed her own inclinations, as the mother had no control over her, and the father was either absent from home or sick. The patient told me, after she entered St. Luke's Hospital, that she had had several severe falls, and that she had been pushed down by her playmates at school, and that after each fall her hip would hurt her more. She said that she was afraid that the splint would be applied again and that she did not come to the Dispensary on that account. Be that as it may, on September 23, 1878, just four years after the patient was discharged as cured, she presented herself at the Dispensary again, when she was examined by Dr. George A. Peters and myself. The thigh was found to be flexed at 75° and adducted at 30°. The measurements were as follows:

Length left leg	28¼	inches
Length right leg	29	"
Circumference of the left thigh at a point six inches below ant. sup. spine of ilium	13⅝	"
Circumference of the right thigh at a point six inches below the ant. sup. spine of ilium	15⅜	"

There was no perceptible motion at the hip-joint, no pain, and there was no evidence of inflammatory action. There was, apparently, an anchylosis of the hip-joint in a very bad position, rendering locomotion without crutches impossible. Under these circumstances it was advised that the patient enter St. Luke's Hospital for the purpose of having Adam's operation performed, in which advice Dr. Peters concurred.

The patient accordingly entered St. Luke's Hospital on January 8, 1879, in the service of the orthopædic surgeon.

The same measurements as those previously recorded are found in the St. Luke's record, and a few days previous to the operation I examined the joint under ether, to assure myself that the condition was not one of pseudo-anchylosis. No movement was perceptible under a not very profound anæsthesia.

On January 28, 1879, with full Listerian precautions, and assisted by Dr. G. H. Wynkoop, I made an incision and passed the saw down to the neck of the femur. After sawing for about thirty minutes, I estimated that the bone was nearly severed, and that it would be safe to make an attempt to fracture the remaining bridge of bone.

I withdrew the saw and made forcible flexion. The limb yielded. After some manipulation, and without the aid of tenotomy, the limb was brought into a good position. The wound was washed out thoroughly with a solution of carbolic acid (1–40) and considerable "sawdust" removed. A complete Lister dressing was applied, without a drainage tube, and the limb was placed in a long extension hip splint, with the lateral screw.

For the following memoranda I am indebted to my assistant at St. Luke's Hospital, Dr. John F. Ridlon.

January 28, 8 P. M.—Complains of pain in hip. ℞ Magend. Sol. *m. v.*

January 29, 7 P. M.—Redressed under spray. There has been a small amount of bloody discharge. Temperature, 100.8°.

February 2.—Temperature has raised from 100° to 101.2°. Redressed at noon under the spray. There has been some serous discharge. No pus.

February 4.—Re-opened under spray. Same conditions as before. Little serous discharge. Temperature (P. M.), 101.2°.

February 8.—Dressing removed under spray. Lister discontinued. Simple dressing substituted, there being only a little serous discharge. Opening closing. Irrigate with 1–40 carbolic wash. Patient placed in prone position and extension of thigh made to overcome a slight flexion of thigh. Temperature, 100.2°.

February 12.—Some puffing at point of incision, which closed two or three days ago; opened and a little brownish serum discharged. Temperature, 101.6°

February 15.—Still little serous discharge; no pus has, as yet, been discharged. Temperature, 101° (P. M.). There was discovered a slight *abduction* of the limb. Straps were passed over the shoulders, from the hip band of apparatus, and by the aid of the lateral screw adduction was successfully performed; opening closed.

February 17.—Limb in good position.

February 19.—Some puffiness again at seat of incision. Opened as before, with some serous discharge, about ℥ ss in quantity.

March 1.—Temperature ranges between 99° and 100°. No pus has been discharged from the opening. Patient is improving in appearance.

March 9.—Temperature normal for several days. Opening has been closed several days. No puffiness or pain.

March 22.—Temperature has not been taken since last entry, patient having done so well. Puffiness and fluctuation again at point of incision, which has remained closed since last entry, the patient having been kept in the recumbent position ever since the operation. Incision made and exit given to about ℥ j of *sero-pus*.

March 22, 3 P. M.—Temperature, 103.5°; 7 P. M. 102°.

March 23.—Afternoon temperature, 101°. The cavity is hyper-distended with 1–40 solution of acid carbolic, and subsequently injected with Bals. Peru. and covered with oakum; about ℥ ss of thin pus is discharged daily.

March 30.—Afternoon temperature, 101°. Has been normal for several days. No pain. The probe does not touch dead bone.

March 31.—Felt chilly last night, vomited early this morning. Had quite a decided chill to-day. Temperature 103°. No cause of trouble apparent, except the pus. Complains of pain at times in thigh and at knee. Cavity and discharge about the same.

April 2.—Temperature down to 99° and 100° again. No bad symptoms.

April 11.—Temperature went up again to 103.5° and went still higher in evening.

April 14.—Temperature again at 99°. There is some tenderness and induration on anterior part of thigh just below Poupart's ligament.

April 20.—Evening temperature, 103°. Good general condition except during time of high temperature.

April 21.—Temperature about normal again. Cavity remains about the same as three weeks ago.

May 13.—For several days a small abscess has been forming a little below the ant. sup. spine of ilium. To-day it opened spontaneously. ℥ i of pus discharged with considerable bloody serum. Injection of the sinus at point of operation, finds its way out through this abscess. The probe passes into old sinus about four inches directly inwards, following the line of original incision, but does not reach bare or necrosed bone. It also passes in another direction inwards and forwards about the same distance. The probe entered the last abscess only a short distance. No dead bone discovered in any of these sinuses.

May 26.—Allowed to get up and move about on crutches. General condition good.

August 15.—General condition has been good. Splint temporarily removed a few days ago on account of excoriations. Splint reapplied to-day. Afternoon temperature 103.2°.

December 12.—Previous attack subsided in a few days. To-day the patient had a severe headache and fever. (No record of temperature appears.) Complains of pain in thigh.

December 15.—A small abscess opened on thigh, and an ounce of healthy pus evacuated.

January 5, 1880.—Discharge has improved and has almost ceased.

March 8.—Very slight discharge. General condition good.

June 10.—General condition excellent. Patient healthy and strong. Walks without an extension splint, sinuses still open.

July 19.—Probe passed in four inches, but no dead bone discovered.

September 15.—Examined by Dr. Geo. A. Peters at my request. No denuded or dead bone could be touched in any of the sinuses.

November 16, 1880.—Discharged improved.

Prior to the discharge of the patient I had on several occasions begged the mother to permit me to enlarge the original incision, and to make a thorough investigation into the condition of the parts. She always refused, and when the patient was discharged, it was with the understanding that if she became worse she was to return for a further operation.

After her discharge from the hospital the patient changed her residence, and though I made repeated efforts to find her, I could not succeed in discovering her. On February 9, 1881, the mother came to the Dispensary again and requested professional attendance. The patient, under the poor care and unrestricted exercise, had become gradually worse. The limb had again become deformed, being flexed and adducted. Sinus after sinus appeared upon the hip, until it looked, at a short distance, as though it had been riddled by buck shot. The probe now reached dead bone, and it appeared as if the shaft was wholly exempt from the necrotic process. At the site of the head of the bone there was very evident necrosis.

Again the mother declined to permit any operative interference, and I reluctantly turned the patient over to Dr. Simeon A. Foster, the out-door visiting surgeon of the Orthopædic Dispensary, who, at my request, kept a record of his visits and watched the patient until her death, which occurred from exhaustion on April 1, 1881, two years and three months after the operation. The mother declined to permit any explorations after death.

The behavior of this patient after the operation is very suggestive. Without going into a detailed analysis of the symptoms presented, it would perhaps be well to give the impressions I received at the time of the operation, and to ascertain, in the absence of post-mortem certainties, if the subsequent erratic course of the case does not confirm these impressions.

As a matter of precaution, it will be remembered, I gave the patient ether a day or two prior to the operation, to determine whether or not actual anchylosis existed. The test at the time satisfied me that true anchylosis did exist, though I did not carry the anæsthesia to a very great degree. When, however, at the time of the operation, the anæsthesia became profound, and the muscles acting upon the hip became thoroughly relaxed, and I had made a nearly complete section of the neck of the femur, I applied a considerable force, with a view of fracturing the remaining bridge of bone. I believe this bridge did not fracture. The motion occurred, I am quite sure, at the joint itself—an incomplete anchylosis being weaker than the unsevered neck. I was struck, in sawing through the neck, with its extreme density. It certainly was healthy bone tissue. Had a false point of motion been established at the place of partial section, there would have been a *sudden* fracture. But, and this excited my suspicions at the time, the yielding was gradual, and I could feel a succession of short "snaps," such as I have felt when breaking up a partially anchylosed knee-joint.

The condition of affairs, then I thought, amounted to this. I had made a partial and nearly complete section of the neck of the femur, and had re-established motion at the formerly diseased articular surfaces. Just how soon the partially severed section would become necrotic under those circumstances I thought, depended upon the depth of the section. In other words, the fate of this section of bone depended upon its blood supply, with a certainty that a violent acute inflammation would occur at the disturbed joint surfaces.

The sequel, I think bears me out in my suspicions, and carries with it a lesson that, I feel, ought to be recorded. Had I been permitted to enlarge the original incision and remove the necrotic bone I think that the result would have been different.

DIASTASIS OF THE TROCHANTER MAJOR, SIMULATING HIP-JOINT DISEASE[1]

THERE are many conditions, which, in some important respects, resemble hip-joint disease. In some instances this resemblance is very close indeed, especially from an objective standpoint. The attitude of the patient, the position of the limb, the limp, the flattening of the nates, the asymmetry of the gluteo-femoral creases, and the modified joint mobility, which occur in true disease may be present in the simulated condition with the subjective element of pain in that region or in those regions where it is most likely to occur in the real disease. If, in a case presenting these features, there be added the element of traumatism as a direct cause of the symptoms, diagnosis may become difficult, and, as experience proves, errors are likely to occur under these circumstances.

The following case presents some very peculiar features and involves a condition which, so far as the writer knows, has not been described in connection with the differential diagnosis of hip-joint disease.

Miss B., a young lady of 17 years of age, of a marked nervous temperament, and a school-teacher by profession, consulted me in June, 1881, presenting for consideration the following history:

About three months before consulting me she was thrown from her horse, falling heavily upon a hard road and striking upon the right hip and side, the principal force being expended upon the trochanter major. Though not rendered unconscious by the shock, the patient can give no adequate idea of her sensation at the time of the accident. She was carried home, suffering a good deal from pain and shock, and placed in bed. She was unable at first to move the limb. It was over two weeks before she attempted to stand and several weeks elapsed before the patient could use the limb in locomotion. She moved about on crutches for awhile, and finally commenced to walk in a peculiar manner, with a decided limp and with pain "in and about the hip," especially after making an effort beyond a few steps. The pain was referred to the trochanter major, to a point behind and above it, and at times there was pain about the knee-joint. This, in brief, was the history given.

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[1] *The Esculapian*, February, 1884.

Before examining the hip-joint in detail I satisfied myself by careful measurements and tests that the limbs were of equal length and that no intra-capsular fracture existed.

I then examined the hip and the surrounding region, inspecting the suspected parts and comparing them with the opposite side. I found—and this was a very marked feature of the case—a very pronounced flattening of the right side of the nates. The right gluteo-femoral crease was slightly higher than the left. There was a slight flexion of the thigh accompanied by slight adduction and rotation inward. The pelvis was tilted upward on the right side, and there was a slight compensatory curve of the spine. The thigh muscles were perceptibly atrophied. In walking the patient limped. Her attitude was very like that of hip-joint disease—in the stage of beginning deformity. Pressure made upon the two trochanters simultaneously in a line toward the mesial plane gave rise to pain; so also did pressure over the hip-joint on the right side. Concussion of the joint by striking the knee forcibly occasioned pain. All these facts, together with the history of traumatism suggested to me that there might be a condition of chronic articular osteitis, as a direct result of the injury, such, for example, as occurred in the case of F. H. P., which I reported in the Archives of Clinical Surgery for June, 1877.[1]

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[1] On Reflex Muscular Contraction and Atrophy in Joint Disease.

Those who are accustomed to making examinations in cases involving the question of the existence of hip-joint disease know, however, that there is nothing more fallacious than the objective signs alone. If there is any chronic inflammation of the joint, especially if there is commencing deformity, there will be sure to be some muscular protection of the articulation, which can not be counterfeited as successfully as are the signs I have just enumerated. I therefore proceeded to make some careful tests as to the "muscular sense" of the suspected articulation.

The patient was placed in the *supine* position upon a hard couch. The various movements of the suspected joint were compared with those of the unaffected side. Flexion of the thigh was very slightly resisted. Adduction and abduction were normal or nearly so. Rotation and circumduction were slightly resisted, and were accompanied by what I thought was a very distinct cartilaginous crepitation at the hip-joint. Placing the patient in the *prone* position and holding the pelvis firmly with the left hand, I tested extension of the thigh. This movement was slightly resisted. So also was rotation of the thigh in and out in this position, and again I obtained the cartilaginous creaking. I again imagined that this crepitation occurred at the hip-joint. But I did not get any reflex muscular protection of the hip, which I would certainly expect to find had there been a condition of chronic articular osteitis. The muscular resistance was variable, as I proved by repeated examinations. These tests led me to think that there was no serious trouble at the hip. But I was at loss to account for the distinct, almost audible crepitation, which I felt had a value beyond the crepitation often found without any perceptible joint change.

Pursuing the examination further, in my effort to get a more decided expression from the patient regarding the peculiar crepitation, and the slight, though easily demonstrated limitation of joint mobility, I again tested rotation of the thigh with the patient in the supine position. On this occasion, however, I placed one hand over the trochanter major, while I performed the movement with the other. To my surprise, the crepitation seemed to be directly under the hand which rested on the trochanter. Following up this clue, I examined the trochanter more closely, and found a false point of motion. I could then produce this crepitation at will by simply making movement at this point. In other words, I found that the trochanteric epiphysis had become separated from the shaft, and the most of the symptoms were explained.

I say "the most of the symptoms." There is no reason apparent to the writer, for example, why there should have been pain at the knee, nor why concussion of the hip-joint should have occasioned pain in this condition. Nor can all the symptoms be explained by the simple diastasis at the epiphyseal line. The girl was very emotional and apprehensive. She had been examined quite frequently, and had been told not only the important and leading symptoms of hip-joint disease, but had been told by several medical men that she had this disease. I do not doubt that the subjective element in this case was greatly influenced by these facts.

The patient was informed as to the nature of the trouble, and was advised to make, so far as she could, an effort to adapt herself to the novel condition of affairs. The apprehension regarding the danger of using the limb, against which she had been warned, was removed. The patient was assured that exercise in moderation would be a benefit. She left me much encouraged. After a month she returned, much improved in every particular, though the false point of motion and the crepitation remained. She walked with a somewhat peculiar, but not notably awkward gait, and had no pain at any point. The movements of the hip-joint gave no evidence of disease or inflammation. She tired easily, but walked without much inconvenience except after prolonged exertion. There was at the last examination no apparent increase in the displacement of the detached epiphysis and the muscles inserted into it have adapted themselves to the new condition of affairs in a very satisfactory manner.

PART III

Club-Foot, Flat-Foot, and Non-Deforming Club-Foot

NON-DEFORMING CLUB-FOOT WITH REMARKS ON ITS PATHOLOGY[1]

Read before the New York Orthopædic Society, February 26, 1885.

THE disabling conditions which are entailed by club-foot are too well known to warrant any extended description. It is not my object on this occasion to dwell upon this point, but to call attention to a condition which, though less conspicuous than true club-foot, is accompanied by many of its annoyances, and in some cases even by its disability; and which I think has not been described or noticed by writers upon either general or orthopædic surgery. At least, I have been unable, after an extended search, to find any such record; and I have also asked those most likely to have information upon the subject, and the result has been entirely negative. It may be well also to say a word in explanation of the name I have given this condition—non-deforming club-foot.[2] It is an apparent solecism, but I think it best expresses my meaning, and certainly it is no worse than "angular curvature," of which we often hear.

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[1] *The Medical Record*, May 23, 1885.

[2] During the discussion of this paper the title I have chosen to designate the condition described met with criticism. No one, however, suggested a better name, unless that used by Dr. Berg is such. In his remarks he referred to "incomplete club-foot." But "incomplete club-foot" does not express my meaning. The club-foot is complete in all its essential particulars. The deformity is so slight, however, that it has heretofore escaped the observation of surgeons and writers on deformity. Hence it is that I retain the title notwithstanding the friendly criticism of my colleagues.

In non-deforming club-foot all the conditions found in certain forms of talipes exist, with the exception of the exaggerated deformity. That is, there is a loss of normal relation between the articulation at the ankle and the muscles which act upon it, involving also in many instances the tarsus, producing a condition which prevents normal flexion at the ankle-joint, and modified mobility, with slight deformity at the tarsal, metatarsal, and phalangeal articulations.

With this state of affairs, we find as a result, varying with the conditions present, actual disability, pain, sometimes very severe, in various parts of the foot, ankle, leg, and even reflected to the lumbar region, and tender and inflamed articular surfaces, especially at the junction of the first metatarsal bone with its phalanx.

If these effects be wanting, we may have only an awkward or peculiar gait associated with painful callosities and corns at various points upon the foot.

The integrity of locomotion depends to a great extent upon a proper degree of flexion and extension at the ankle-joint, and upon a normal condition of the plantar arch and plantar tissues. As, however, the conditions which will be considered on this occasion depend upon a modified flexion at the ankle-joint, and a contracted state of the plantar tissues with their sequelæ, we will omit a discussion of other conditions except as they may throw light upon the subject. Nor is it intended to discuss at length the mechanism of the ankle and the various bones of the foot. We have certain and easily demonstrated clinical conditions to account for, and an extensive dissertation upon mechanics is not necessary.

But there are a few points which may help us, and I will briefly allude to them.

First, as to the ankle. Its function, except in extreme extension, is that of a plain hinge-joint. In the conditions we have to deal with, flexion and extension are the only movements to be considered. Extension of the foot, in the adult, is limited at about 135°, or 45° more than a right angle, using the long axis of the tibia as the plane of measurement. Flexion stops at about 70°, or 20° less than a right angle (see Fig. 1). The position of the foot in standing upon an even surface, with the knee in full extension, is about 90°. The amount of flexion and extension varies in different individuals, but these figures,

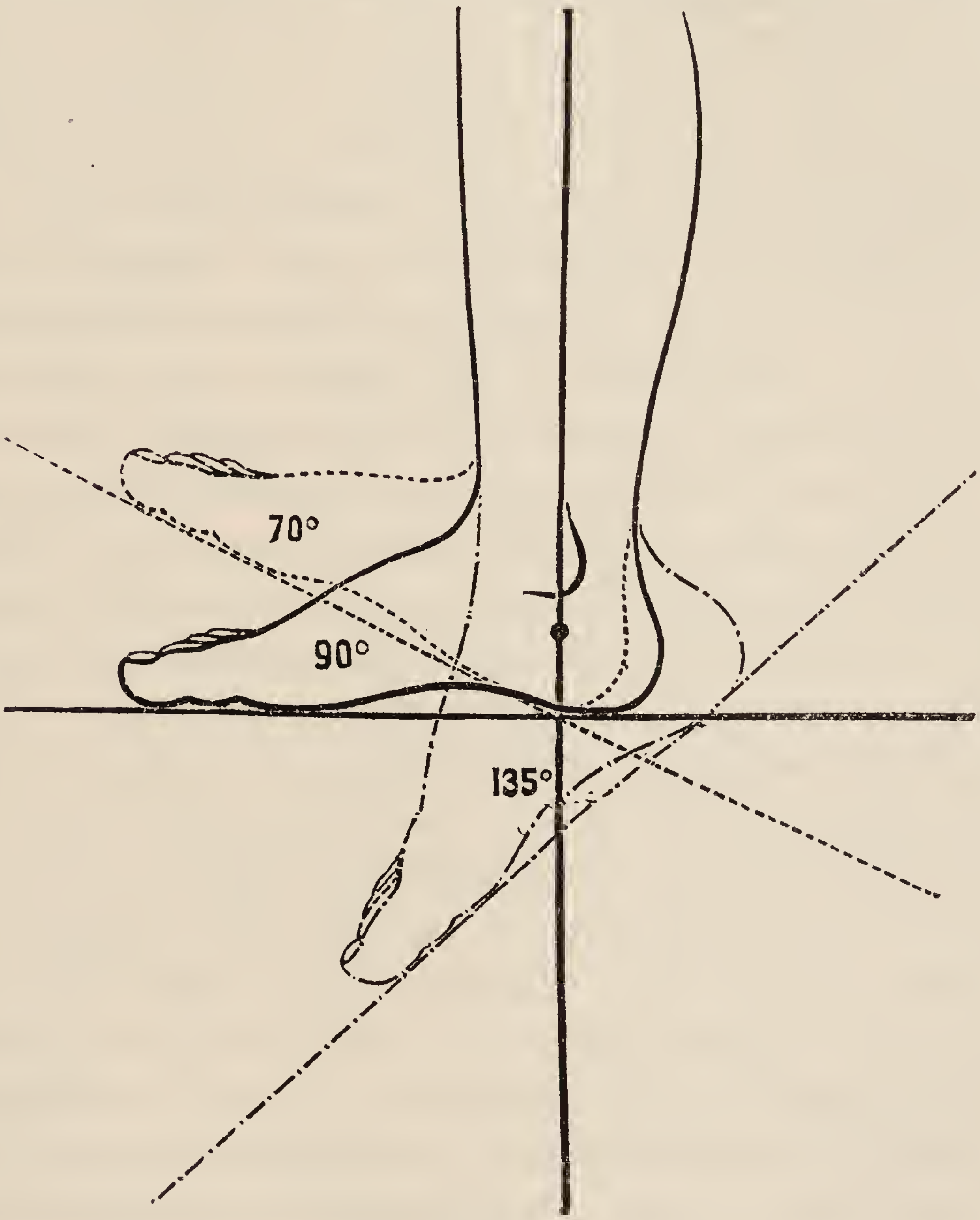

Fig. 1

based upon actual experiment and measurement, represent, I think, the average of normal movement in the living adult subject.

We should remember the general structure and function of the tarsus and metatarsus in sustaining the weight of the body, and especially the relation of the plantar tissues to the arch formed by these bones, and the articulations between them. Grossly speaking, the plantar tissues represent the bow-string of a bow, and they, with the articulations in the arch above, furnish the basis upon which elasticity is furnished to the arch. With each step the arch is "sprung" slightly in normal locomotion; but if we have a contraction of the plantar tissues, this elasticity is destroyed or interfered with, and the "ball of the foot" is drawn toward the heel to a greater or less extent. The os calcis does not move forward toward the "ball of the foot," especially if the gastrocnemius muscle is shortened. In calcaneus, where the gastrocnemius muscle is paralyzed, the plantar tissues contract and draw the heel toward the metatarsus and phalanges. In each case the least resisting part is drawn toward the more fixed part, and modifications of the tarsal arch of different characters are seen. In each case also the tarsal arch is exaggerated; and, while we can have a calcaneo-valgus with a contracted or shortened plantar fascia, it is only, in my experience, when the gastrocnemius muscle is paralyzed or weakened. The *inner* part of the foot is, so to speak, the *varus part*, and, with the exception above noted, a contraction of the plantar tissues results in a varus with an adduction of the tarsus and metatarsus.

The outer part of the foot is not so likely to be shortened antero-posteriorly as is the inner part. The outer part of the foot in normal locomotion touches the floor at each step, and is deprived of elasticity in the same sense as the plantar arch. It is intended for direct contact, and affords an unyielding support. It becomes affected secondarily in club-foot, when pes cavus results.

Practically speaking, with the exception noted, we can divide the foot longitudinally, as applied to the deformities of club-foot, at the mid-tarsal and mid-metatarsal region, just as we can divide the foot transversely at the mediotarsal joint. As before stated, we do not expect to find a shortened plantar fascia in valgus, nor do we look for an elongated condition of the plantar tissues in varus. If the plantar arch gives way, valgus results. If the arch is exaggerated with the exception noted, we have one of two deformities. First, the antero-posterior contraction in the plantar region draws the ball of the foot nearer the heel, and brings the metatarsal bones more nearly in a line with the long axis of the tibia, producing that position so frequently seen as the accessory of pes equinus, with little or no varus; or, secondly, we find a true varus with the metatarsal bones thrown into an adducted position, *i.e.*, toward the mesial plane. In exaggerated cases both of these conditions may exist. In non-deforming equinus, of the type now under consideration, there is generally a slight exaggeration of the tarsal arch, with little or no adduction of the tarsus and metatarsus. The deformity at the tarsus, in short, is antero-posterior, and not lateral; and while we may have and frequently see a non-deforming club-foot presenting as a valgoid state, it is not of this condition that I wish to speak.

Depending upon the portion of the plantar tissues affected, we have as a result of the condition now under consideration a shortening of the inner half of the foot. The inner part of the foot contracts, antero-posteriorly principally, with the result of making the plantar arch noticeably hollow just posterior to the junction of the first metatarsal bone with its phalanx. (See Figs. 2 and 3.)

The two inner metatarsal bones assume a position more nearly approximating a vertical line, and the whole of the inner half of the distal part of the foot is thrown out of normal relation to the rest of the foot and the body.

The three outer metatarsal bones are not normal under these circumstances. Their relations to the other parts of the foot are, however, considerably less disturbed than are the two inner ones.

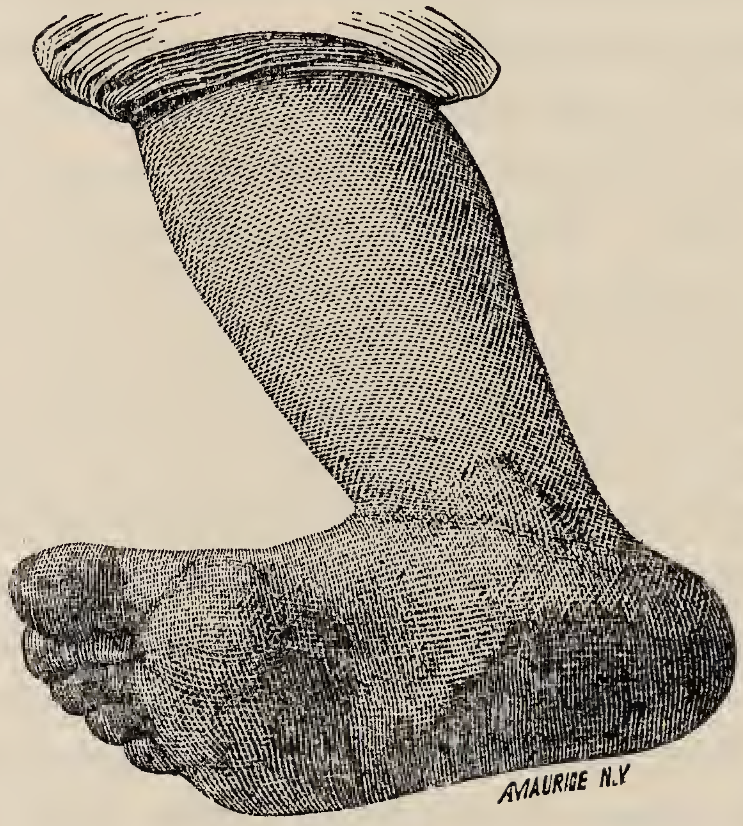

FIG. 2

And now, what happens as to the muscular relations? The flexors of the ankle-joint proper are unable to perform the act of flexion. Owing to the plantar resistance or the gastrocnemius resistance, or both, and the altered relation of the metatarsal bones and the tarsus to the ankle-joint, the distal part of the foot passes beyond the complete control of the ankle-flexors. A peculiar feature of non-deforming club-foot now presents. The extensors of the toes act as assistant flexors of the ankle, and in doing this it is necessary that the extensors should complete their function as such before they can aid the crippled ankle-joint flexors. Hence, all those who have non-deforming club-foot with plantar or gastrocnemius contractions, when asked to flex the ankle, put the toes in extreme extension in the effort to accomplish ankle-joint flexion.[1]

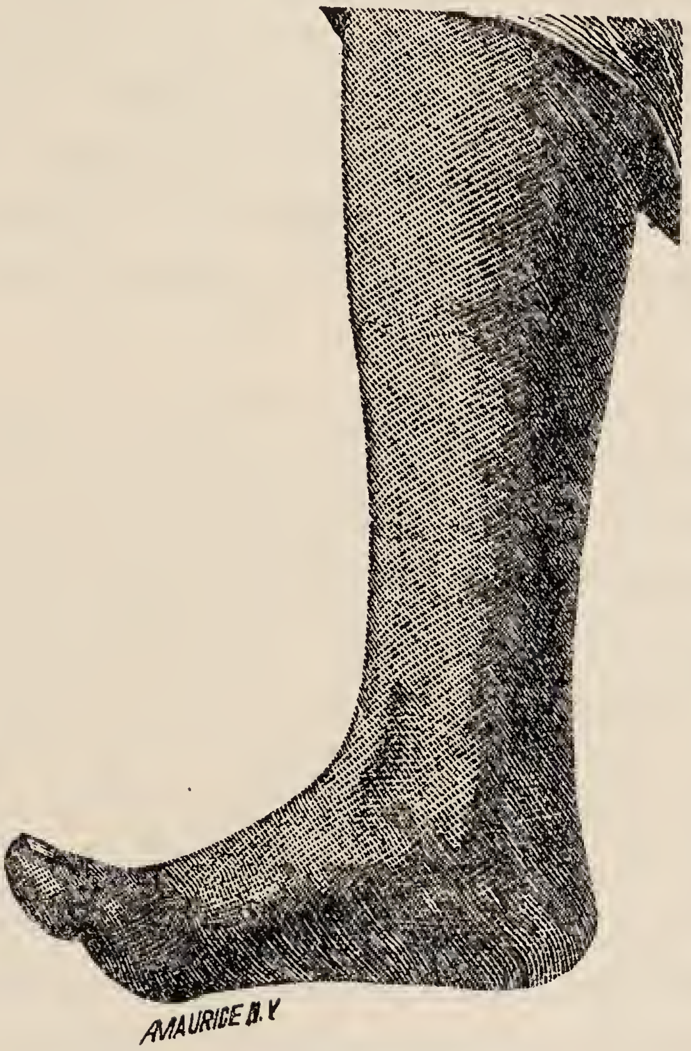

FIG. 3

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[1] (See again Figs. 2 and 3.) The toes are thrown into extreme extension, and the tibialis anticus stands out prominently, as seen in Fig. 3, and yet the patient could not flex the ankle sufficiently to raise the "ball of the foot" to the 90° line. So also in Fig. 2, the plantar depression and the prominent and calloused "ball" is very evident. The patient from whom these engravings were made, came to the Orthopædic Dispensary suffering from severe pain in the thigh, and was limping badly. Only one limb was

Not only, then, are the inconveniences and disabilities of non-deforming club-foot due to contractions of the muscular tissue and fasciæ, with subsequent changes in ligaments and bone—thus putting the complicated foot- and ankle-mechanism at a disadvantage—but also to a very important loss of elasticity at the tarsus. When this elasticity is lost completely in simple flat-foot, the gait and the disability are easily recognized. When the elasticity is modified by a shortened state of the plantar tissues and the arch is exaggerated, a different disability is present, the principal features of which I shall soon point out.

It is unusual to find a gastrocnemius shortening without some changes in the plantar tissues also. When, however, as sometimes happens, the gastrocnemius shortening is the principal factor in non-deforming club-foot, flexion may be arrested at any point about the right-angled position. Most frequently the foot stops at 90°, or a little more or less. An awkward gait is apt to occur with gastrocnemius shortening and slight plantar trouble; while pain, "gouty big-toe joints," and disability are apt to occur when the plantar troubles are in excess.

If the condition which I have tried to describe exists, while the patient is aware that something is radically wrong, he does not recognize the cause. The trouble, not enough in many instances to warrant belief that a surgeon

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affected. It was supposed by the physician who sent the patient to the institution that she was suffering from hip-joint disease. All her symptoms arose from the slight non-deforming club-foot, which escaped the notice of the entire assistant staff of the dispensary. The patient was exhibited at the meeting after the reading of this paper. It is this condition which constitutes true non-deforming club-foot. The patient was unaware of her condition; many physicians examined her without discovering her true trouble, and it required a close examination and tests to demonstrate the slight deformity upon which depended the pain and disability.

could be of service, comes on gradually, and is frequently a process of slow growth. It becomes a sort of "second nature" to the individual, and he comments on his disability or awkward and peculiar gait; he curses his gouty toe, or condemns his shoemaker, or applies iodine or cocaine to his painful ankle or tarsus.

Perhaps he takes a long course of "anti-rheumatic" or "anti-gout" remedies under the direction of intelligent and reputable physicians. He probably spends considerable money in obtaining temporary relief at the chiropodists, and looks in vain for the, as yet, undiscovered "anatomical" shoemaker. But the sought-for relief does not come.

Let us suppose that, under these circumstances, a person afflicted with non-deforming club-foot presents for examination. He removes his shoes and stockings, and presents his feet for inspection, occupying a seat upon a chair which is near that of the surgeon. Under these circumstances the foot falls at once, and naturally into a position of pes equinus, and all that one notices, unless his attention is especially directed to some other point, is the prominent "big-toe joint" with its swollen and tender surfaces, or the painful corns at different points. The real elements of the trouble are lost in the natural "toe drop" of the position taken by the patient. If, on the other hand, the patient is really disabled by "an exacerbation of the gouty symptoms," and is confined to his bed, again the equinus position of the foot is apt to deceive, and the attention of the surgeon is directed, not to the cause of the trouble, but to an effect. Rest and local applications, with or without constitutional remedies, are generally prescribed; and the rest produces relief. The patient goes about again after a few days, only, however, to inflict an injury upon the vulnerable tissues, and to suffer another relapse; when again the "gouty toe" receives attention.

Now let us see what a critical examination of a patient in this condition shows. After examining the foot as to sensitiveness, etc., and inspecting the inflamed big-toe joint, ask the patient to stand up. He will probably be able to stand erect with his feet upon the floor, in the "heel and toe" position; for the weight of the body acts as a flexor and the elasticity of the tarsal arch is not wholly lost. There is no apparent deformity, no great variation from the normal. The feet are, perhaps, not quite straight, but then the patient will tell you that it has always been so; as if this made it right. He can walk across the floor without apparent trouble. Ask him now to stand squarely against the wall, with the knees in full extension; ask him to throw the weight of the body upon one limb, while he attempts to flex the opposite ankle-joint. He will be surprised to find that he has little or no flexion. It is then that the toe extensors come into bold relief as aids to ankle-joint flexion. Test both ankles in this way. Perhaps both will show modified flexion; more generally only one will be affected to a marked degree; the other being slightly crippled only. In some cases a marked plantar resistance, accompanied only by a slight gastrocnemius contraction, permits tolerably good ankle flexion, but not enough to raise the "ball of the foot" far from the floor. Still better, put the patient in the recumbent position, and ask him to flex the ankle with the knee straight. He cannot bring the "ball of the foot" into the normal plane of the heel, the former dropping from one fourth to three fourths of an inch below the latter. The resistance cannot be overcome by voluntary muscular effort, though the patient concentrate all his energies in this direction. Again the toe extensors come prominently into play, and, in some cases, the phalanges assume a position nearly at right angles with the long axis of the first metatarsal bone. In this position try manual pressure. You find that the tissues, both plantar and post-tibial, form a resistance which you cannot overcome. You only render the resisting tissues more tense by your efforts. You cannot do more if you give an anæsthetic. The conditions found are those of true club-foot; the contraction and malposition are permanent. The sequelæ are there, and the condition is one that, in many instances, demands treatment.

Non-deforming club-foot may exist, and does frequently exist, without the attending evils mentioned in the foregoing remarks. I have tried in this sketch to outline a typical case, accompanied by more than simple inconvenience. There are a large number of cases, in young and growing girls especially, where inability to flex the ankle does not produce more than a slight inconvenience, or an awkward carriage or a peculiar gait. I shall refer to these cases farther on.

It will be seen that non-deforming club-foot of the class which I shall dwell principally upon on this occasion approximates the conditions found in equinus or equinovarus. There is another condition which time does not permit me to discuss, viz., simple flat-foot, that presents a valgoid condition. But here in certain conditions there is a shortened gastrocnemius with relaxed plantar tissues and a flattened arch.

Nor can we stop to consider inflammatory flat-foot. It has certain signs and symptoms which render any remarks on differential diagnosis unnecessary, for there is no primary local inflammation in the class of cases I wish to describe.

The inflammation in true non-deforming club-foot is the effect of the altered articular relations and the long-continued and abnormal pressure. The inflammation, happily, with its attendant pain, swelling, corns, and callosities, speedily ceases when the cause is removed, and the parts are restored to an approximately normal position and relation. There is only one exception, perhaps, to this rule regarding primary inflammation, and that is in chronic sprain, when we have, in certain instances, a condition which I have ventured to name traumatic non-deforming club-foot.

Non-deforming club-foot may occur at any age. I have seen it in infancy, when doubtless its etiology is the same as that of congenital club-foot. It occurs rarely as an acquired condition in children, though often the "good result" of many surgeons after tenotomy and treatment leaves behind it a condition like that I have attempted to describe. It is seen very frequently during the period of second growth—the adolescent period—in both sexes, when, at that particular age, there are not apt to be many important sequelæ. It usually does not reach its full development until adult life and full growth is attained.

It occurs more frequently in the female sex, and *when looked for*, is found sometimes in rapidly growing girls, and especially in those whose growth has been apparently arrested before the average height is reached. And what is very important, and to a certain extent remarkable is this: *it is found very often associated with true rotatory lateral curvature of the spine.*

The discovery of the co-existence of non-deforming club-foot with true lateral curvature was the result of what proved to be a logical conclusion from the study of certain muscular conditions seen in the true club-foot of children affected with unilateral arrest of development from the, as yet, obscure lesion which occurs in the motor tract of the brain. I have seen many cases of this character, and, after examining a case of this kind with Dr. Berg, the electro-therapeutist of the Orthopædic Dispensary and Hospital, in 1881, I remarked to both Dr. Ketch and Dr. Berg, "The muscular conditions found in this boy's foot remind me of the muscular conditions found in true lateral curvature"; and I further said that I would examine my cases of lateral curvature more carefully and find out the muscular conditions at the ankle-joint. Soon after this I was called to see a patient with true lateral curvature and marked rotation in the mid-dorsal region, Miss C. S——, eighteen years of age, who had been placed under my care by Dr. T. G. Thomas. After inspecting the curves and modifying the apparatus, I asked permission to examine the ankles. The patient had on either side a typical non-deforming club-foot. The gastrocnemii were short, the plantar tissues were contracted, the prominent toes were there, callosities were present; indeed, all the signs and all the conditions that go to make up a true case of non-deforming club-foot were found. The peculiar gait and "rheumatic pains" of the patient were accounted for, and the slight "ankle weakness" which existed in this young lady was explained. Since that time I have examined many cases of true scoliosis, and while some of them escape, there is in a large number a modified ankle-joint flexion due to muscular changes and muscular contracture. And I have examined, as opportunity presented, young girls especially, who have other troubles than scoliosis. It sometimes happens that in a case where there is no apparent cause, the non-deforming club-foot exists, though the percentage is very small as compared with the scoliotic subjects. It does not follow, therefore, that all scoliotic girls should necessarily have ankle-joint complications, but fully, or over one half of my cases present non-deforming club-foot as a condition that accompanies true scoliosis. A portion of my notes on this subject have been mislaid, and I cannot, I am sorry to say, give accurate figures. One thing, however, may be regarded as certain; and that is, that to the mammary atrophy, the shortened leg, and the other trophic signs of true lateral curvature may be added the non-deforming club-foot, which is more frequently present, I think, than either the unilateral shortening or the mammary atrophy.

This, to say the least, coincidence is somewhat remarkable, and may yet throw some light upon the etiology of true scoliosis by directing our attention toward the central nervous system in our efforts to find the ultimate cause of this, as yet, obscure condition.

The conditions found in non-deforming club-foot of the type especially that accompanies the lateral curvature are certainly suggestive. There is no paralysis, the electrical reactions of the muscles are normal and sensation is unimpaired. There are, however, sometimes marked unilateral differences, especially at the upper and lower extremities, both as to length and circumference. There seems to be, in many cases, a unilateral difference of growth. Even if one leg is shorter than the other, muscular power, with the tests I have applied, seems to be normal. So far as the ankle and the non-deforming club-foot are concerned, it is found that as soon as the mechanical relations are restored the patient is able to perform all the movements normally. The reflexes are apt, especially the patellar tendon reflex, to be exaggerated, though in some instances this is not so. Again, it frequently happens that the non-deforming club-foot is not present in the shorter limb, but in the longer one, as if the bone had, so to speak, outgrown the muscles (see remarks under Class 4). There is no evidence of motor-spinal complications (eliminating, of course, the cases referred to under Class 1). It seems to me that some cause, congenital or acquired, has produced, in some cases, a unilateral, in others a bilateral misdirection of growth rather than a typical arrest of development, which, in some instances, shows itself at the neck (true torticollis); at the spine (true scoliosis); or, at the feet (non-deforming club-foot). In non-deforming club-foot the changes are not always apparent in early life. Like true torticollis, the changes are not apparent till growth develops them. But as growth occurs, and especially as the second or adolescent period of growth appears, slight and often unnoticed defects come into prominence, and when full growth is attained, as in the conditions found in non-deforming club-foot, they become more apparent, and they are frequently disabling. In torticollis these changes are apparent in early life; in scoliosis they are noticed most frequently in adolescence; in non-deforming club-foot they are often entirely unnoticed in childhood, become apparent at puberty, and are burdensome later in life. If we accept the lessons taught us by neurology we must look, not to a peripheral lesion for our cause, nor to habit, nor to the spinal cord, but to the, as yet, little, pathologically speaking, understood motor tract of the brain for the primary lesion which causes the slowly progressive changes in the muscles, and, secondarily, in the bones, joints, and fibrous tissues.

Aside from the reasons and inferences given here, I have other grounds for my belief, as expressed above, and the best way to express them is to classify the subject as far as is possible from a pathological standpoint.

We may have a condition of non-deforming club-foot from five different causes: (1) Non-deforming club-foot seen after polio-myelitis anterior; (2) non-deforming club-foot which follows simple and uncomplicated malposition, habit, etc.; (3) non-deforming club-foot produced by traumatism, sprains, etc.; (4) non-deforming club-foot found after the infectious diseases of childhood, especially diphtheria and scarlet fever; (5) the non-deforming club-foot due, as I believe, to some remote trophic disturbance, and seen quite frequently co-existing with true lateral curvature.

As briefly and as concisely as possible I will refer to these different conditions:

1. Non-deforming club-foot seen after polio-myelitis anterior. As long ago as 1865, I noticed quite frequently that when both lower extremities had been paralyzed and one had apparently entirely recovered; or in some cases, where the history pointed to only a unilateral paralysis, as a sequel of polio-myelitis anterior, that at the ankle-joint and at the tarsus of the so-called "well foot," certain abnormalities would appear. In other words, and as a matter of illustration, a certain amount of varus or equino-varus would be present. Since that time I have seen many instances of this kind, and I have frequently been consulted by my patients, in this condition, for advice regarding the "well foot." Indeed, I have come to consider it almost exceptional, after an analysis of many cases, to find a foot free from contraction, and perfectly normal after recovery from polio-myelitis in childhood and affecting one or both lower extremities. As a rule, however, the inconvenience produced by the non-deforming club-foot, under these circumstances, is so slight compared with the great disability of the affected side or limb that little or no notice is taken of it either by the patient or surgeon. But the conditions found at the ankle and tarsus of the limb which apparently has recovered have often been sufficient to produce just the troubles

I have pointed out; and as before remarked, I have benefited several patients greatly by treating the apparently slight contractions found at the tarsus and calf. In many cases the amount of non-deforming club-foot, under these circumstances would be considered a great annoyance and burden if the disabled limb were a normal one in all other respects.

Under these circumstances we have a well-recognized pathological condition to account for our non-deforming club-foot. The acute inflammation in the anterior horns of the spinal cord is the cause of this condition, and the imperfect recovery leaves, primarily, a slightly disturbed muscular, and, secondarily, osseous and ligamentous changes which make a complete picture of non-deforming club-foot. In some of these cases I have found deficient power in the tibialis anticus muscle, but not generally. In the majority of cases, there is no real loss of power that can be defined or measured with the faradic current, and unlike the non-deforming club-foot that I have attempted to describe in the foregoing pages, it frequently appears in childhood as soon, indeed, in some cases, as the more marked deformity and atrophy at the more extensively disabled limb. It, like the trophic non-deforming club-foot of Class 5, becomes more annoying in adult life unless it receives early attention; and I make it my rule now to treat these contractures, however slight, in children or in even older persons.

I was inclined at first to attribute what I have since called for want of a better name, the trophic non-deforming club-foot to a slight and unobserved attack of poliomyelitis, occurring early and remaining undiscovered until adult life brought the defect to the notice of the victim. And I am not certain that, in some cases, this is not true. But the absence of early symptoms, the frequent limitation of the trouble to the tarsal tissues principally, the normal reaction of the muscles, the presence of an exaggerated tendon reflex at the knee, and the absence of specific atrophy, or rather the presence, in many cases, of large, well-formed, and strong limbs, seems to exclude polio-myelitis from the larger group of cases, nor can I find in any of my cases any history of an illness like that which marks polio-myelitis. There is in none of my cases any history which leads me to think of the spinal cord; on the contrary, the clinical picture points to another motor tract, viz., that of the brain.

Where a marked equinus or equino-varus ensues after polio-myelitis, and frequently after years of treatment, the best result often obtained leaves an ankle-joint that cannot be flexed to a right angle. Of course voluntary power over the flexors is gone, and the patient in attempting to walk, cannot bring the heel squarely upon the floor. This very seriously interferes with locomotion, especially if the quadriceps extensor muscle is impaired by the central lesion. We have here another form of club-foot which needs attention, and which can be greatly relieved without tenotomy. Indeed, I have been very greatly surprised in several cases to find considerable muscular power developed by overcoming the contractions without tenotomy, giving the patient the opportunity of flexing the ankle while standing upright, using the weight of the body as a mechanical aid to its production.

2. Non-deforming club-foot produced by malposition, habit, etc. It will be noticed frequently that patients who have been for a long time in the recumbent position, or who have a shortened lower extremity, present a slight amount of equinus. In these cases there is not much tarsal shortening, and in all of them the deformity is overcome generally very easily by manipulation, or what is better,

use of the limb and joints. There is no organic trouble, or complicating inflammation, or polio-myelitis back of the contraction, and as the trouble is functional, restoration soon follows if the patient has full functional use of the limb. Habit, or continued malposition, is not, I am assured, the cause of true non-deforming club-foot; of that kind, for example, which is associated with the lateral curvature. We can, again, for example, have a lateral curvature due to habit, but not the true scoliosis that is accompanied by rotation. The non-deforming club-foot of habit is not the club-foot of scoliosis, where simple habit, I am sure, does not enter into the etiological factors at all.

A great deal has been said by both the medical profession and the laity regarding the subject of high-heeled shoes as a factor in the production of various ills. As regards this element, I may say at once that all the cases I have seen of non-deforming club-foot, due to obscure causes, have worn low-heeled boots, and have rarely worn tight shoes. High heels afford sometimes a temporary relief to the victims of non-deforming club-foot. But the advantage gained, as to the shortened gastrocnemius, is soon lost by crowding the tarsus and foot into a very uncomfortable and painful position, and high heels, if tried, are soon discarded. To those who have plantar contractions alone, these high heels produce, as one of my patients said to me, "perfect misery." Happily, the "anatomical" shoemakers are using low heels and broad soles. These are the shoes sought for by those who suffer from non-deforming club-foot.

So far as the habit of wearing high heels is concerned, it may be dismissed as a factor in the production of obscure cases of non-deforming club-foot that I have attempted to describe.

3. Non-deforming club-foot produced by simple traumatism, sprains, etc. I need not stop here to dwell upon the conditions found in chronic sprain. Suffice it to say that, as a result of the injury, seconded by the malposition and reinforced by adventitious inflammatory products, there results a condition of affairs which, after the acute symptoms subside, leaves the foot in a position of slight deformity. This deformity is almost always an equinus, with or without some slight lateral malposition, and, as is well known, complete disability frequently results.

Such a case presented at my office on May 24, 1882, having been referred to me by Dr. A. L. Loomis.

Mr. S——, aged sixty-five, residing in Central New York, sustained a severe sprain of the right ankle in July, 1881. He was treated by the usual methods and by competent men, with a not unusual result. He came into my office on crutches, which he had been using ever since the acute symptoms had subsided, about nine months. He had suffered very much, and had tried almost everything, he said, and had now come to New York with the idea of having the foot amputated if he did not find relief.

It is needless to transcribe my notes in detail. I need only say that it was a bad case of chronic sprain. The foot was in a position of extension, with slight tarsal contraction. The patient was unable to stand upon the foot, or to bear any weight upon it; first, because the shortened post-tibial muscles and tissues prevented any motion whatever in the direction of flexion; and, secondly, because any attempt in that direction gave great pain. He was a man of great strength of character, and made every effort to flex the ankle, but without any success.

The indications seemed very plain; I knew that if I could elongate the shortened gastrocnemius and lengthen even slightly the plantar tissues, thus affording even slight ankle-joint flexion, the patient would have normal joint surfaces to use, and not the abnormally sensitive ones that were then in contact. I asked him if he would consent to an experiment that would probably hurt somewhat. I stated if he objected to the pain I might occasion, I could give him an anæsthetic, but told him if he remained under my care the experiment must be tried. He told me to do what I saw fit, and that he could bear any pain I might inflict. I put the foot in an extension shoe,[1] and applied traction to the extent of the patient's endurance. He walked alone and unaided that day. Eight applications of the extension shoe, covering as many days, relieved this man so completely that he returned home using only a cane. He soon discarded even that.

One more illustration of the benefit of the extension shoe in the traumatic non-deforming club-foot.

A young lady, Miss P——, aged nineteen, consulted me on May 2, 1884. She had sprained her ankle in getting out of a sleigh, in December of the previous year. After a prolonged treatment she was told that she was well, and that she must walk. She tried to walk and failed. Gradually she became able to walk a block, or, at most, two blocks. This slight effort occasioned pain of an excruciating character at the medio-tarsal joint. I saw her five months after the accident, and she was still unable to walk more than two blocks. Her general health was beginning to suffer and her friends were very anxious about her condition.

Upon examination of the foot, I found a shortened gastrocnemius and a contracted plantar fascia. The ankle could not be flexed beyond ninety degrees, and any effort to carry it further gave rise to pain. The patient could not voluntarily flex the ankle above ninety degrees. With the other ankle flexion was normal, and there had been no trouble with the affected joint prior to the injury.

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[1] I have described the "extension shoe" in an article on "Traction in the Treatment of Club-Foot," in the New York *Medical Record* for November 23, 1878. The present article will soon be followed by another, giving the author's more recent experience with its use, and with detailed instructions as to its management in both non-deforming and true club-foot.

Nine applications of the extension shoe, covering two weeks, completely relieved this patient. She has had no return of the trouble since.

4. The non-deforming club-foot found after the infectious diseases of childhood, especially diphtheria and scarlet fever. Dr. A. Jacobi on a recent occasion (*vide* the New York *Medical Journal*, August 9, 1884) calls attention to a condition which may throw light upon certain cases of non-deforming club-foot. His remarks are upon "The Relation of Over-nutrition After the Acute Fevers of Childhood to Bone Disease." After mentioning the fact that children not only seemed, but really were taller, Dr. Jacobi says, "the body became taller by an elongation of the bones; the bones grew by a rapid proliferation about the cartilage."

I have seen two cases recently which seem to substantiate this statement, and which further seem to demonstrate that during this rapid growth of the bones the muscles do not keep pace with them. The result is a considerable non-deforming equinus in each case. It is several months since the acute infectious process subsided, and yet there remains a slight but easily demonstrated non-deforming club-foot, and more, I am quite sure, than that which results from simple recumbency. And I have seen several cases where scarlet fever and diphtheria left behind as their legacy a lesion producing a slight hemiplegia, and trophic disturbances resulting in a non-deforming club-foot. Indeed, it is well worth while for us to investigate the relation of these infectious diseases as etiological factors in the production of such conditions as those found in non-deforming club-foot, both in children and in adults.

It may be in place to remark here that in a large number of young girls that I have examined for lateral curvature, and in whose history an early attack of diphtheria or scarlet fever was the only important or tangible etiological factor, I have found an easily demonstrated non-deforming club-foot. If, as may be the case, the etiology of true scoliosis and the accompanying ankle-joint contraction are the same, we may find that the infectious diseases of childhood play an important rôle in their production.

5. The non-deforming club-foot due, I believe, to some remote trophic disturbance, and seen quite frequently co-existing with true lateral curvature. The first case of this kind of which I can furnish any notes was seen in consultation with Dr. M. Putnam Jacobi, on April 22, 1880. From exhaustive and complete notes furnished by Dr. Jacobi I extract the following:

Miss A. B—— was a tolerably healthy child. Between eleven and twelve developed insufficiency of the internal recti muscles; used glasses for a few months, and occasionally a few years later. Has only recently discarded them. Was the eighth child in a family where only eighteen to twenty-four months intervened between any two births.

Father healthy; lost one sister from epilepsy. Mother healthy and of a long-lived family. The brothers and sisters were all more or less delicate; one sister has insufficiency of the internal recti muscles.

Patient menstruated at fourteen; was extremely irregular until twenty; was subject to nervous and sick headaches. At the age of twenty, the first attempt was made to re-establish menses by leeches to cervix uteri. This was successfully accomplished in May, 1879, and continued regularly throughout the summer. In September, 1879, patient, being in the mountains, sprained her foot. Sprain supposed to be slight, but two months later attempt at walking continued to cause great pain. Patient remained confined to her room for eight months without setting the foot to the ground. During this time occasional paroxysms of neuralgiform pain in foot, radiating from inner border near scaphoid metatarsal articulation. Pressure at this point always extremely painful. Conviction gradually entertained that patient was suffering from an hysterical arthralgia supervening on a sprain which had merely been an exciting cause. It was, nevertheless, impossible to induce patient, though a very sensible girl, to use the foot, for each attempt was followed by pain often extremely severe. The general health of the patient was perfect—the first time that this had been the case since the age of twelve.

In the spring of 1880 Dr. Shaffer was asked to see her. He noted the marked malposition of the foot and partial extension and adduction, and attributed the pain to the retraction of the plantar fascia. Dr. Shaffer advised the use of a mechanical shoe, which should gradually stretch the fascia, and recommended that the patient commence to walk at once. This she did with marked relief. She went to Europe, and walked well. While there she heard news which produced quite a profound shock, and various hysterical symptoms manifested themselves. After a varied experience she returned to her home and is now quite well.

Throughout Dr. Jacobi's notes there are various comments upon the hysterical elements as presented by the patient. These I recognized when I examined the patient, and I gave them their full value. I am sure Dr. Jacobi quite agrees with me in attributing the condition of the patient at the time of our consultation to the retracted plantar fascia and shortened gastrocnemius, which rendered the foot very vulnerable. The sprain, which was slight, occurred in a foot which was in a condition of non-deforming equinus. The hysterical condition of the patient only added to the difficulties of the diagnosis and obscured the real condition. On February 20th, of this year, we again met in consultation and found our patient was still suffering from a shortened plantar fascia and gastrocnemius. Tested in standing position the patient could not flex the ankle-joint. She has a true non-deforming club-foot, with the danger before her, as has frequently happened, of sprains and trouble at the ankle upon slight provocation.

The case is also very instructive as showing how very vulnerable a joint and foot may become with slight contractures, and how long a person may be disabled by a sprain under these circumstances. The pain produced in these cases is sometimes very severe. In a neurotic girl, such as Miss B—— certainly was, the pain was very greatly exaggerated.

In this case there are none of the ordinary causes which can be relied upon to account for the conditions found at the ankle-joint and tarsus. There is no history which furnishes any clue. It does not come under any of the previous classifications. There is no evidence of a spinal cord lesion; nor is there any evidence of a local peripheral lesion. The contracture exists; it is permanent, and it failed to yield under an anæsthetic. Hence we may safely assume that it was not hysterical, however much the hysterical diatheses may have exaggerated the symptoms.

On December 9, 1881, I was consulted by Miss C——. She came to ask my opinion regarding her right foot and ankle. She experienced great pain in walking, and was frequently actually disabled by the pain, which was principally located at the scaphoid bone and in the plantar tissues. The extent of her walking was only one or two blocks. Her exercise was consequently very limited, and she spent most of her time when out-of-doors in her carriage. She was very liable to sprain her ankle upon slight provocation, and after one of these sprains would frequently spend a month upon her couch. She was in excellent general health, and was, when her ankle would permit, a keen enjoyer of out-door sports. There was nothing in a hereditary sense that indicated any trouble, nor was the early history of the patient suggestive, save that she always remembered having had a peculiar walk from about fifteen years of age; and that while her ankle had troubled her, she did not experience any serious inconvenience, except during the three years prior to the time she consulted me.

Upon inspection of the foot there was nothing in its appearance to account for the trouble. There was nothing to indicate any articular inflammation. A high instep was noticeable, and the dorsal surface of the toes and the ball of the foot were disfigured by corns. There was only one point of tenderness on pressure and that was at the scaphoid bone. The plantar tissues were also very sensitive. The calf was large and well shaped, and the muscles were firm and solid. Upon comparing the afflicted member with its mate no gross differences were perceptible. Both feet, dropping into gentle extension as the patient sat upon a high office-chair, seemed normal. The patient informed me that the shoemaker made her right shoe upon a special last, as the instep was higher and the foot slightly shorter. The limbs were approximately of the same length; and the measurement of the calves showed that the right was slightly smaller than the left—one-eighth of an inch.

The presence of corns led me to suggest that she had been wearing tight shoes. She informed me that she had spent ‘‘nearly a fortune’’ in shoes and upon chiropodists. She could not wear high-heeled boots; she had tried them, and they gave her extreme pain. She had consulted several surgeons in this city and in Europe, and nearly all had accused her of high heels and tight shoes, and some had prescribed a particular shoemaker, but she had obtained no relief. I saw by the tone and manner of my patient I had better give up that line of investigation.

In examining further still with the feet in extension, as the patient sat before me, I found another tender point just anterior to the external malleolus. There was neither heat nor redness, however, and she was unaware that tenderness existed at that point until I discovered it. I began to think the patient might be hysterical. But further investigation led me to discard this as wholly untenable.

Pursuing my investigation further, I commenced to test the ankle-joints as to mobility. I recalled Dr. M. P. Jacobi's case, and asked the patient to attempt voluntary flexion in the standing posture. She was entirely unable to flex the right ankle; the left one was normally flexible. In attempting to flex the right ankle, the "ball of the foot" remained as if glued to the floor. Strong manual effort on my part, with the patient in the recumbent position, failed to accomplish flexion beyond 90^{c}. The plantar tissues were tense; the foot by actual measurement was three-eighths of an inch shorter than its mate, the test being made in the standing position, tracing the outline of the feet carefully upon paper.

The main difficulty in recognizing this condition, as in all similar conditions, arose from the fact that the examination was made with the patient in the sitting position; which means practically with the feet in the quinus position. It was only when joint motion was fully tested that the real cause was apparent.

The young lady had very strong muscles: on the affected side, the muscles seemed almost too strong to think of anything save tenotomy as a remedy. But I had succeeded in lengthening the gastrocnemius one inch and a half with the extension shoe, in a case of equino-varus occurring in my service at St. Luke's Hospital, in a boy of sixteen, and I advised the patient to permit a trial of the extension shoe before we resorted to any operative procedure. She consented. One week later I applied the traction force with my apparatus. The first application, though painful, gave great relief. Twenty applications, covering six weeks in time, gave perfect relief, and there has been no return of the trouble. Ankle-joint flexion, though not normal, is sufficient to enable the patient to walk comfortably and without pain, and by this process the foot was lengthened three eighths of an inch. The plantar tissues yielded as well as the tough gastrocnemius.

I might add to these cases if necessary. Time does not permit their recital, however, and I must pass on to consider a subdivision of this class, having, I believe, the same etiology as the foregoing where the conditions are somewhat different.

6. Non-deforming club-foot found in adults, especially with almost normal gastrocnemius action, but with shortened plantar tissues producing principally a changed relation of the metatarsal bones.

In two cases of this class that I have seen there was a loss of power in the tibialis anticus muscle. Most frequently there is no loss of power at all, and the muscles all respond normally to the faradic tests.

Miss N——, aged twenty-one years, a resident of New York City, came to me in October, 1884, with a letter of introduction from Dr. N. A. Roberts, of this city.

The patient's hereditary history presented nothing that would be likely to throw light upon our subject. There was no history of rheumatism or gout in the family, nor did the patient have any evidence of trouble of this nature.

She could assign no cause for her trouble. When about fourteen she commenced to have pain in her foot. This was soon followed by a swelling at the "big-toe joint." The pain and swelling increased, and she was soon confined to the house, and was unable to walk for months. She consulted several physicians, who ascribed her trouble to one of three causes, viz.: gout, rheumatism, or tight shoes with high heels. Under advice she took much medicine, and went repeatedly to different shoemakers and chiropodists. No permanent relief followed. The traumatism occasioned by even a short walk brought back her disability. Her general health began to suffer as a consequence of her inability to take exercise.

Her condition when I saw her was as follows: There was a very considerable enlargement at the junction of the first metatarsal bone with its phalanx, which was painful to the slightest touch. The skin was very red and tense. There was no particular pain or tenderness elsewhere. The patient was not at all apprehensive regarding the parts, except as pain was inflicted by pressure. Upon examining the ankle-joint I found that normal flexion was impossible. The foot could be flexed only to a right angle. But there was only slight gastrocnemius resistance. The real resistance existed in a markedly shortened plantar fascia, which rendered the metatarsal bones more vertical than normal. There was no atrophy present, and the muscle responded normally to the faradic current. The patient walked with difficulty and with pain.

The first application of the extension shoe gave great relief. The foot was lengthened one quarter inch by the first application. After the second application of the traction force there was complete relief, and the patient now considers herself well; though the traction is kept up occasionally.

Mrs. M—— was sent to me on November 24, 1884, by Dr. E. C. Seguin. Dr. Seguin, in his letter of introduction, states that after a very careful examination he could exclude any constitutional cause as contributing to the condition presented by the patient.

The history of the patient shows that there was no perceptible hereditary influence at work. She had suffered during childhood from only two diseases, viz.: pertussis and rubeola. At fifteen she was fully matured, and had been well all her life.

At an early age it was noticed that she had a peculiar walk. As a little girl her friends used to comment upon it, and then the patient remembers that her ankles when she was a child gave her more or less trouble. At eighteen years of age her feet became more troublesome, and as she advanced in life they became still more so. She never wore tight shoes or high heels. About three years ago the "big-toe joint" on either foot became very troublesome. Pain was experienced at that point with a "cramped feeling" at the plantar arch. Walking became very irksome. The patient says she was conscious of restraint, though she could not tell why. The "big-toe joint" became more prominent and more inflamed. Last autumn she became so disabled that she could not walk over one block without suffering very severe pain. Chiropodists were consulted, with temporary relief, and different shoemakers were tried, with uniform disappointment.

The conditions found upon examination were so like those recorded in the preceding case that repetition seems unnecessary. The slightly shortened gastrocnemii, with the very tense and contracted plantar tissues, made the distal ends of the first and second metatarsal bones, the point where much of the weight of the body fell during the act of walking, more vertical than normal. In this instance both feet were affected.

After the second application of the extension-shoe, the relief was very marked. Traction alone had made the foot very perceptibly longer and narrower, and the patient enjoys to-day entire immunity from her "gouty big-toe joints."

Further duplication of this class of cases seems unnecessary. One word regarding "gout." I have seen nine cases of non-deforming club-foot where the principal trouble has been at the "big-toe joints," the favorite seat of gouty pain and swellings. In almost every case, except the last one (Dr. Seguin's), gout or rheumatism has been diagnosticated by prominent medical men. The removal of all unfavorable symptoms by traction proves one of two things: either these were true cases of gout which were relieved, as to the foot symptoms, by the means used, or they were pseudo-gout caused by abnormal pressure upon the exposed articular surfaces. I think it likely that, in some cases, both elements were at work, and that the elongation of the foot, coupled with the new articular relations thereby resulting, has contributed to the result. I am even led by my experience to say that many cases of true chronic gout at the point mentioned may be greatly relieved by the means employed in these recorded cases.

One word, however, before closing my remarks. The celebrated mare Maud S. presents almost the same condition that I have attempted to describe as occurring in the adult and adolescent. From a recent issue of the New York *Tribune* I copy the following remarks, made by Mr. Bonner in describing the present condition of this celebrated trotter. He says, after some general remarks: "You will observe that only the imprint of her toes is visible. The heels scarcely touch the ground. She is literally walking on her toes. . . . Maud S. has been troubled with severe corns." Mr. Bonner, in a long and scientific way, dwells upon these and other defects in his mare and prescribes for her condition. It seemed to me worth while to stop a moment and notice this very accurate description of the condition I have called to your attention, though applied to a horse rather than to the human being, especially so as my search for medical facts bearing on the subject has been fruitless. And yet there are many persons to-day who are practically walking on their toes, and who, like Maud S., have corns and other disagreeable sequelæ which, I am happy to say, are very easily relieved.

NOTE BY THE AUTHOR

(September, 1923)

The fact that Maud S. had a "Shortened Gastrocnemius" is probably one reason of her great speed. I have never examined the feet of professional runners and jumpers—but I have critically examined the feet of a good many adolescent athletes of both sexes,—amateurs, school and college boys and girls. And I have proven to my satisfaction—that the proficient dancer, the expert skater, the rapid sprinter have slightly shortened gastrocnemii. The pedestrian—the long distance walker, the high jumper, have normal ankle-joint motion. The reason is obvious. The quick recovery after the foot touches the floor is favored by the shortened muscle just as the normal ankle gives a greater spring to the joint as the body rises.

THE USE OF TRACTION IN THE TREATMENT OF CLUB-FOOT[1]

Read before the New York Academy of Medicine, February 3, 1887.

Over twelve years ago I felt that there was room for improvement in the then existing forms of apparatus for the mechanical treatment of club-foot, and, in connection with my service at the New York Orthopædic Dispensary and Hospital, commenced a series of experiments bearing upon this subject, and based upon a study of the mechanism of the ankle and tarsus.

The result of these experiments was published in the *Medical Record* for November 23, 1878, under the title "Traction in the Treatment of Club-Foot." In this article, which may be called a provisional report, I called attention to some of the defects in the conventional form of club-foot apparatus, and described one form of traction apparatus, viz., the antero-posterior (see Figs. 7, 8, 9, *et seq.*), which was devised for overcoming the contractions of uncomplicated talipes equinus. The simple lateral shoe or "pusher," with its hinged lever and screw (see Fig. 22), was also described, the latter being used to overcome the so-called lateral deformities of equino-varus prior to the use of the antero-posterior traction apparatus.

Since the publication of this provisional paper I have continued my investigations, and have made many improvements in the apparatus first described. The antero-posterior traction-shoe has been made much more efficient, and perhaps nearly perfect. The simple "lateral pusher" did not meet all of the indications presented in the compound deformity known as equino-varus, and it has been discarded in the treatment of this condition excepting in early infancy, and then it is used as a preliminary splint, the antero-posterior or lateral traction apparatus being used subsequently.

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[1] *New York Medical Journal*, March 5 and 12, 1887.

This simple "lateral pusher," however, with its hinged lever and screw, suggested the right line of investigation and introduced, I believe, the important *pushing* principle into the mechanical treatment of talipes.[1] Following up the clew it gave, I have, from time to time, added other mechanical pushing and traction elements to it until I have practically perfected the lateral-traction apparatus, which meets quite perfectly the indications presented in the compound deformities of club-foot combining the elements of equinus with either varus or valgus.

In the article already referred to I ventured to point out some of the defects in the mechanism of the conventional club-foot apparatus, the principal one being the difficulty of controlling the heel, which, in the conventional form of apparatus, is held in place, more or less insecurely, by the Scarpa heel-cup, and the retaining strap, which passes from the heel-cup over the neck of the astragalus. All surgeons who have seen and treated many cases of club-foot will at once appreciate this difficulty of controlling the heel with the apparatus ordinarily employed. I did not, when I wrote my provisional essay upon this subject, fully appreciate all the mechanical points involved, though I felt that I had discovered the correct principle of treatment. Further investigation and study enable me to present my theme in a much more tangible shape, and to accompany a description of my apparatus with, so far as I know, a few novel demonstrations of the movements at the ankle and tarsus, which movements we ought to imitate and even exaggerate with the mechanism devised to relieve the deformities of equinus and equino-varus; for it is only these varieties of club-foot that will be considered on this occasion.

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[1] I have searched in vain for a description of an apparatus which fulfills the indications presented by this "lateral pusher." It has a purely lateral action, the hinge being placed at or near the malleolus, and the force is applied to the concave side of the deformity, pushing the entire foot toward the normal position.

I propose to call attention, first, to some points in the mechanism of the normal foot, and to afterward describe the apparatus I have devised to overcome the deformities of talipes.

In Fig. 1 let A B E represent a line drawn from the head of the fibula through the ankle-joint to the sole of the foot, B representing the center of antero-posterior motion at the ankle-joint. C E D represents the sole of the foot, C the heel, and D the toes. The diagram is supposed to present a side-view of the leg and foot, with the foot at right angles with the leg, as in standing upright; a position, conventionally speaking, midway between flexion and extension of the ankle-joint.

In Fig. 2 we have another diagram, similarly lettered, with the ankle in extension. The line A B is the same as in Fig. 1, but at B (the center of antero-posterior motion at the ankle-joint) this line becomes deflected posteriorly on its way to E, the line C E D forming an angle of about 45° with the line A B, the position of full extension of the ankle-joint.

In Fig. 3 we have still another diagram, lettered as in

Figs. 1 and 2, but with the ankle in full flexion. In this figure, at B, the line A B is deflected anteriorly at B on the course to E, the line C E D forming, in normal flexion of the ankle-joint, an angle of about 20° with the line A B.

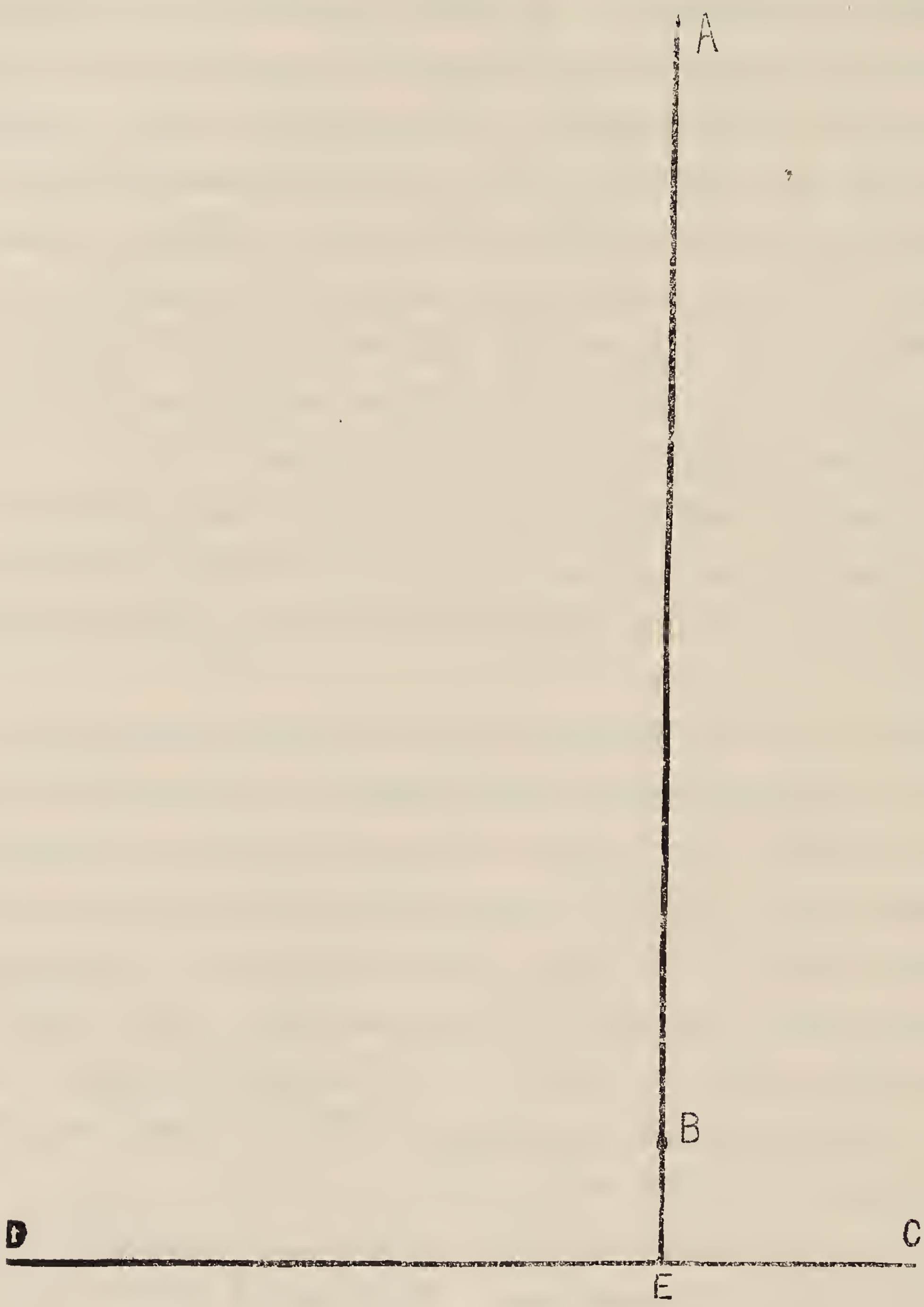

Fig. 1

In these three diagrams, as in the normal foot, there is only one center of antero-posterior rotation, and this is at

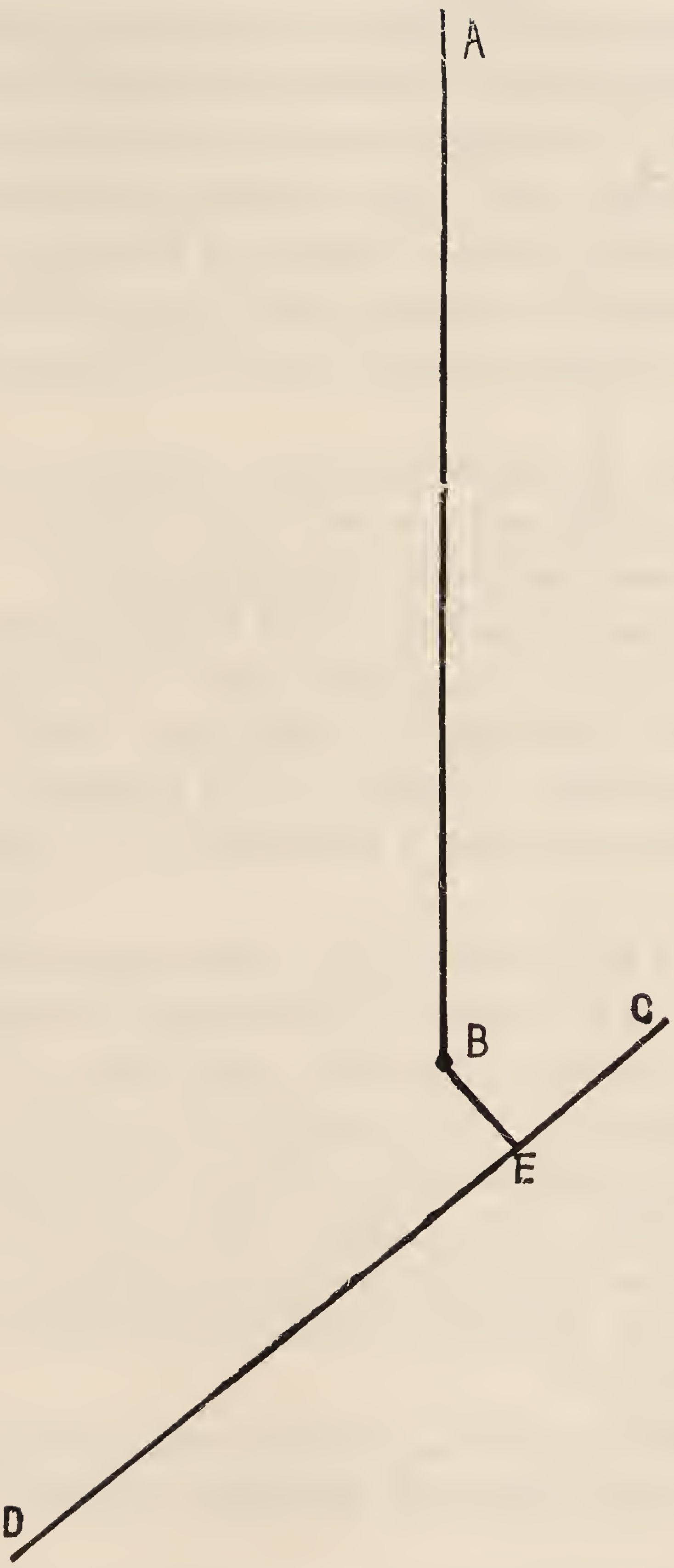

FIG. 2

the point B.[1] It is not at the ankle joint, but considerably below it, and in all our efforts to change a position of permanent extension, due to muscular or fibrous resistance, to that of flexion, we ought to construct the apparatus devised for this purpose with this definitely located center of motion properly placed. Indeed, it is well to exaggerate the force applied by placing this joint of the mechanism lower down than the normal center of motion at the ankle-joint.

This center of antero-posterior rotation at the ankle-joint being recognized, we will now combine these three diagrams in one and study the combination.

In Fig. 4 the vertical line A B I corresponds with A B E in Fig. 1; C I D corresponds with C E D, Fig. 1; A B K and E K F in this diagram correspond with A B E and C E D, respectively, of Fig. 2; A B L and G L H of this diagram correspond with A B E and C E D, respectively, of Fig. 3.

In short, A B K and E K F represent extension of the ankle-joint. A B I and C I D represent the position when the foot is between extension and flexion, the so-called normal position, or that occupied in standing erect with the foot fairly upon the floor. A B L and G L H represent extreme flexion. All of these three positions have one common center of antero-posterior rotation, viz., B, just as there is only one center of antero-posterior rotation at the ankle-joint.

We may now apply the principle demonstrated by these diagrams to the deformity of talipes equinus. The ankle is in, we will suppose, full extension, and the heel is represented by the letter E, in Fig. 4. The problem is to force the heel, E, to take the position G, and to carry the toes

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[1] "The axis of rotation of the curved superior surface of the astragalus passes through the most fixed part of the bone, viz., the tarsal canal, touching the outer malleolus, but passing below the inner, which does not descend so low."—Frank Baker, in Wood's *Handbook of the Medical Sciences*, article "Ankle Joint."

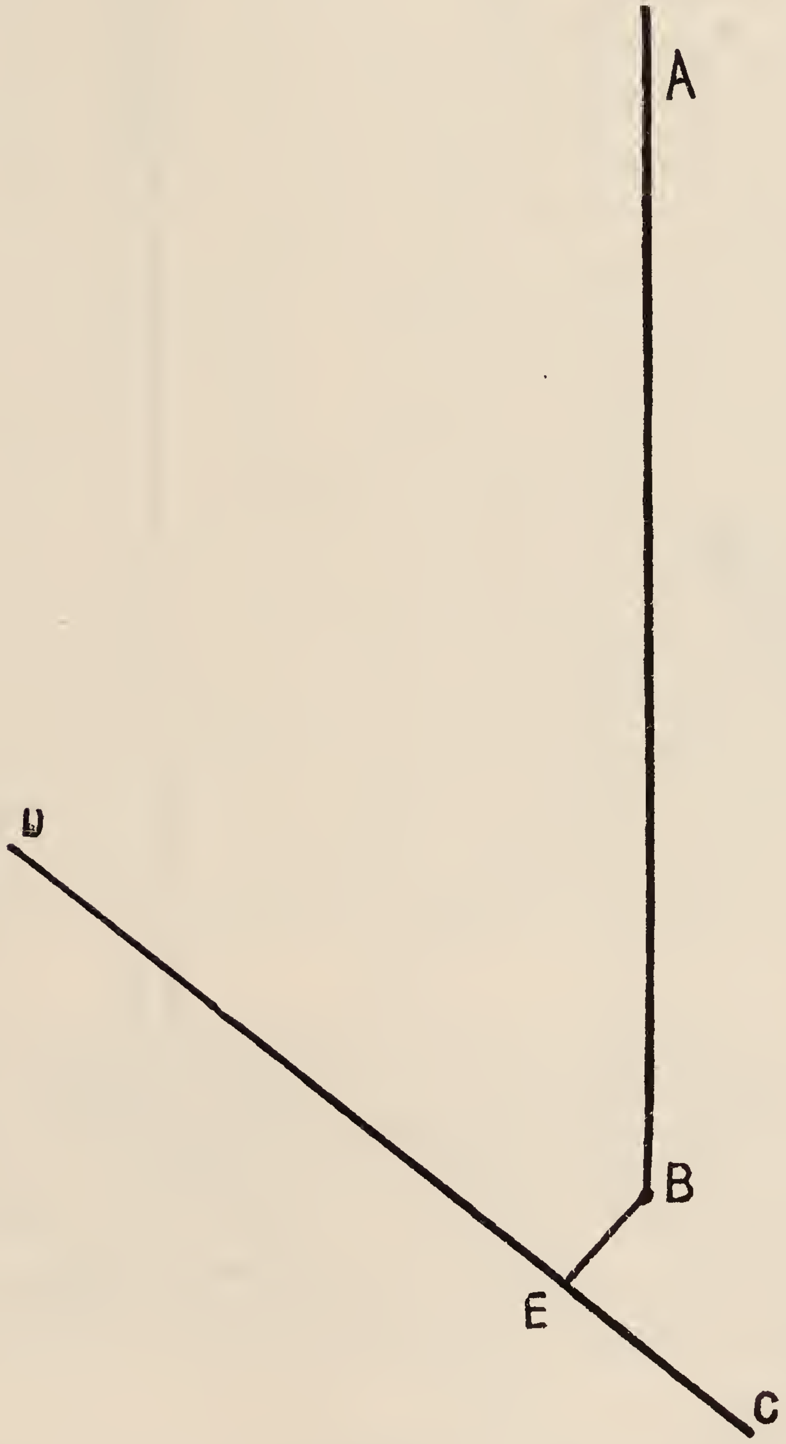

FIG. 3

from the position F to that of H. The heel, rotating around its center of motion B, must follow the curved arrow E C G, and the toes must also follow the curved arrow F D H.

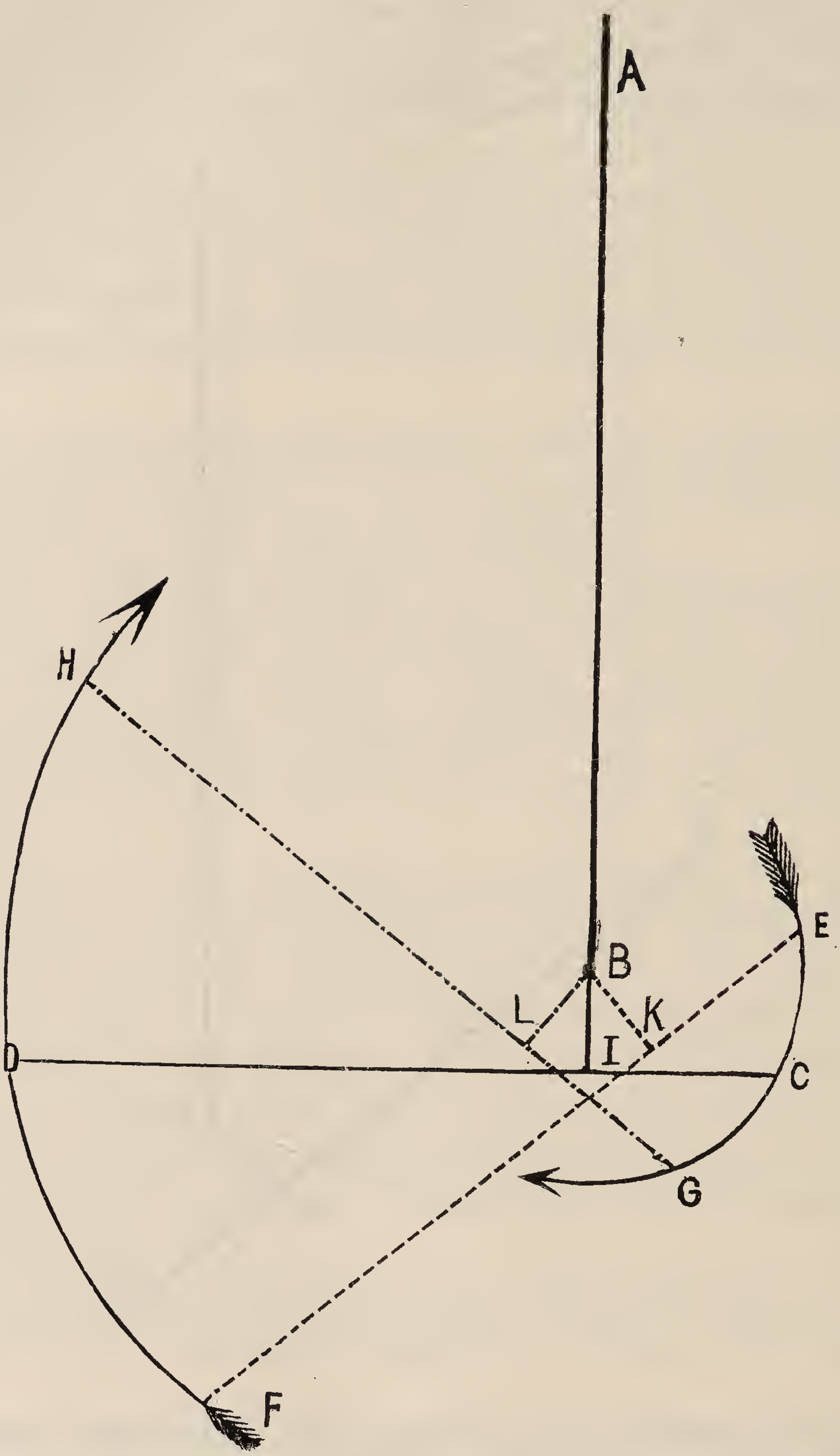

Fig. 4

It naturally follows that the arrows E C G and F D H represent segments of a circle, each having a common center B, the latter having a longer radius.

This fundamental position being recognized, we can now study the direction of the curved heel and toe arrows, and attempt to apply the results of our study to the antero-posterior deformity known as talipes equinus.

Taking the point opposite the center of motion, B, as the plane of measurement, the direction of the heel arrow is *downward* and *forward*. Taking the same center and plane again, the direction of the toe arrow is *upward* and *forward*. When the right-angled position is reached, the heel arrow still goes *downward* and *forward*, being carried below the plane of the standing position, and the toe arrow has a direction *upward* and *backward*, or toward the vertical line (A B, Fig. 4), as in normal flexion of the foot.[1]

Any apparatus devised for the relief of the conditions found in talipes equinus ought to carry out these indications, and present movements based upon a correct interpretation of normal antero-posterior ankle-joint rotation.

It may be said that the conventional forms of club-foot apparatus present the movements here decribed. Many of them do, but no adequate means is taken to make the deformed foot, restrained by muscular contraction, follow the direction taken by the curved arrows; and, for reasons now to be stated, they can not be made to accomplish this end.

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[1] When the "right-angled position" (Fig. 1) is reached in overcoming the deformity of talipes equinus, the deformity is practically reduced. The antero-posterior traction-shoe is so arranged, however, that any desired position of flexion may be obtained, the traction-bar being made long enough to forcibly carry the heel *downward* and *forward* to any desired extent, thus forcing the toes *upward* and *backward* toward the tibia when the "right-angled position" is passed.

As I stated in the paper already referred to, when criticizing the conventional method of retaining the heel in the heel-cup of the ordinary forms of apparatus devised for the treatment of club-foot, "the point at which the retaining force and counter-pressure is made" (the neck of the astragalus) " must also rotate around the pivotal point" (the center of motion indicated by B in all the foregoing diagrams).

In Fig. 5 let A represent the center of motion at the ankle-joint. A H represents extension of the ankle-joint; A I, the right-angled position; A J, flexion. When the heel rotates around the center of motion, A, and goes *downward* and *forward* (curved arrow B C), the toes pass *upward* and *forward* (curved arrow F G), the neck of the astragalus passes from H^1 (extension) to I^1 (right-angled position) to J^1 (flexion), and it (the neck of the astragalus) follows its own curved arrow, D E, the direction of which is *upward* and *backward*. In short, the heel rotates *downward*, and the neck of the astragalus rotates *upward* around the transverse center of antero-posterior motion at the ankle-joint.

It becomes plain, therefore, that the neck of the astragalus, being tied down, as in the conventional form of apparatus, can not rotate *upward*, and, if this movement is blocked, the heel can not go *downward*. When flexion is attempted in the ordinary forms of apparatus, with the retaining heel-strap applied, there are these insurmountable mechanical impediments that effectually prevent the foot from following the mechanical law of antero-posterior rotation which I have attempted to describe.

If this heel-strap which passes over the neck of the astragalus is loosened to permit the movements indicated in the conventional form of apparatus, all control over the foot is lost.

And this brings us to an important point that ought to be considered. The center of motion of the ankle-joint is

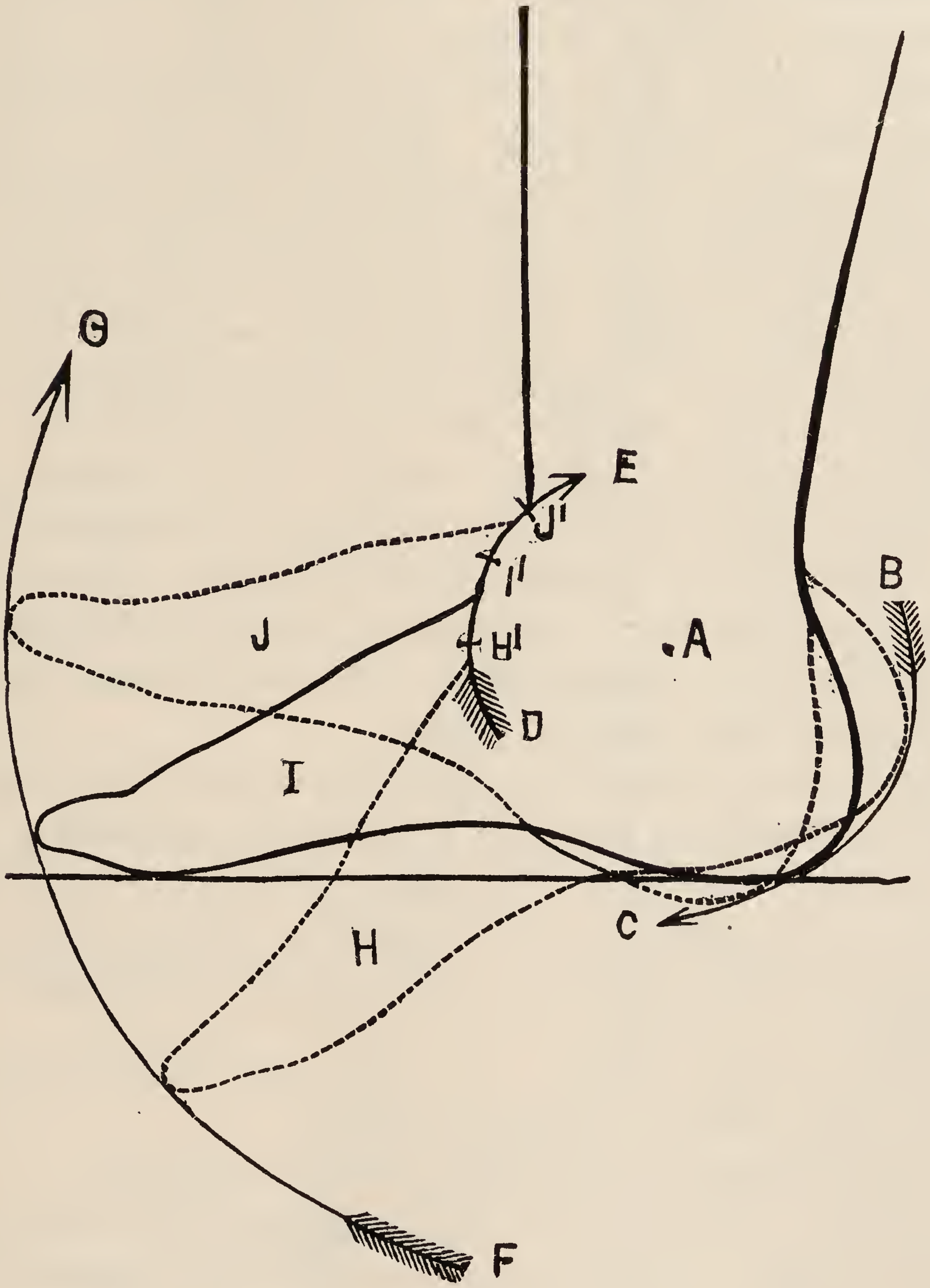

FIG. 5

below the malleoli. While this anatomical center of antero-posterior rotation of the ankle can not be changed, and we can make the artificial center in the apparatus correspond with it, the restraining force necessary to produce forcible flexion of the ankle must be applied at the *rotating astragalar neck.* If we could pass an iron bar transversely through the foot at the center of antero-posterior motion of the ankle, the restraining astragalus strap would be unnecessary. Even if this could be done, the foot would not follow the apparatus with strong contractions holding it in a deformed position unless the foot were securely fastened to the foot-plate of the apparatus by some such means as the traction heel-strap of my apparatus (soon to be described), and an adequate force applied to bring the foot into the desired position. It should be remembered that we are not, in treating the conditions found in club-foot, applying a mechanism to a foot with normal antero-posterior motion. There are strong contractions to be overcome, and the normal movements should be *exaggerated*, in order to exert the proper *traction* upon the resisting tissues.

In the conventional form of club-foot shoe the rotation of the astragalus is blocked by the faulty mechanism, antero-posterior rotation of the ankle-joint is prevented, the heel is not controlled by the Scarpa heel-cup, and no adequate force is applied to carry the heel *downward* and *forward.*

There is another fault no less important in the mechanism of the ordinary form of apparatus.

I will attempt to explain it by another diagram:

In Fig. 6, A B I and C I D correspond with A B E and C E D of Fig. 1 when the foot is at the right-angled position. A B K and E K F correspond with A B E and C E D of Fig. 2 when the foot is in extension. In the conventional form of apparatus the flexion force is applied in the direction of the arrow, R S, which is mechanically incorrect and opposed to the movements sought for. The heel should move *downward* and *forward*, the toes should go *upward* and *forward*, the head and neck of the astragalus

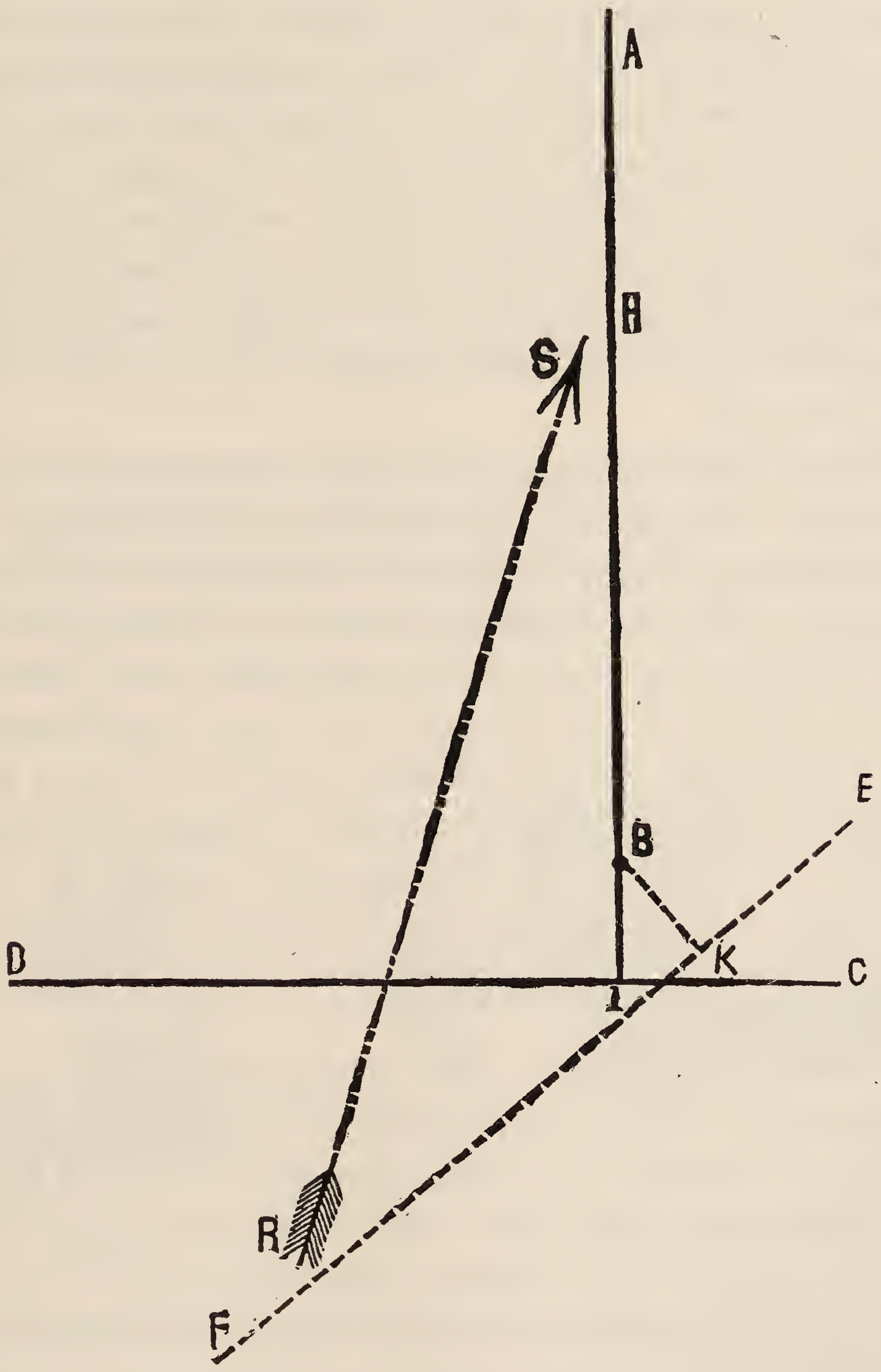

FIG. 6

should pass *upward* and *backward*, in the first movement of talipes equinus. Any force, be it elastic or otherwise, applied in the direction of the arrow, R S, in talipes equinus, antagonizes these movements, and favors the *upward* and *backward* movement of the heel, especially if a restraining astragalar strap is used to block the upward rotation of the astragalus. When the right-angled position of the foot is reached, the movement indicated by the arrow, R S, is correct, if the heel is brought downward and forward by some traction force, and allowance is made for the rotating astragalar neck. But when the right-angled position is reached, the difficult feature of the deformity is removed, and the favorable result is practically assured.

Still another fault exists in the conventional form of apparatus. No provision is made in the foot-plate of the apparatus for the heel as it passes *downward* and *forward*. As soon as the right-angled position is passed, the heel drops below, just as the toes pass above, the plane presented by the sole of the foot as it rests upon the floor. This is shown in the curved heel arrows in Figs. 4 and 5. As soon as the right-angled position is passed, the further rotation of the heel is permanently obstructed in the conventional form of apparatus by the foot-plate of the apparatus, which should be provided with an opening for the descending heel.

It should be the object of treatment in talipes equinus to restore the normal movements of the ankle, if possible; certainly to produce a certain amount of flexion beyond the right-angled position. The apparatus about to be described permits any desired angle of flexion to be obtained, and the apparatus is especially constructed with this end in view.

These are the principal points of criticism to be made upon the conventional forms of apparatus obtainable in the shops of the instrument-makers. There are others I might urge were there time or inclination. Those mentioned are the most important, and demand the attention of all who are interested in the treatment of club-foot.

Having pointed out that which I consider radically defective in the apparatus ordinarily used for the treatment of club-foot, and which I was taught to use and did use for years, I come now to a description of what I have termed the "traction apparatus," which, I think, not only meets the mechanical indications mentioned, but permits the application of sufficient force to overcome many of the deformities presented without the intervention of tenotomy.

As the diagrams I have presented bear only or principally upon the indications for the treatment of antero-posterior deformities, with plantar or post-tibial contraction, or both, I will describe the "antero-posterior traction apparatus" first, and call attention to the "lateral-traction shoe" afterward.

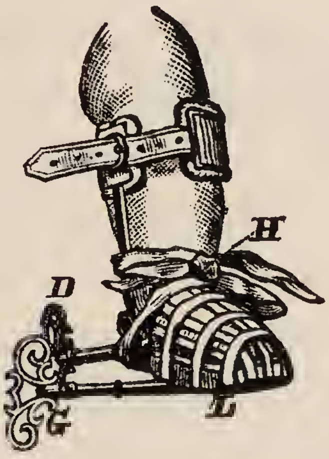

FIG. 7

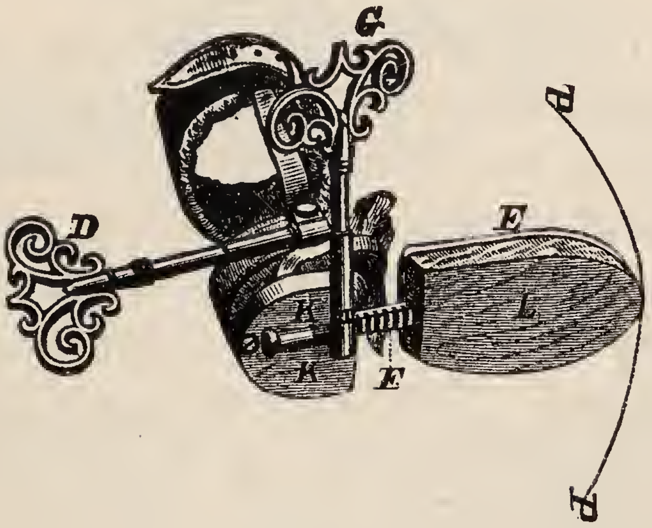

FIG. 8

In the paper already referred to I described this antero-posterior traction-shoe. I then attached the apparatus to the foot by means of adhesive plaster. The apparatus with its adhesive-plaster attachment, as originally used, is shown in Fig. 7.

Figs. 8 and 9 were also used to illustrate the apparatus. I have long since discarded the use of adhesive plaster in making traction for a much more ready and convenient method, as will be seen by a comparison of these engravings with the engravings that illustrate the apparatus as used at present.

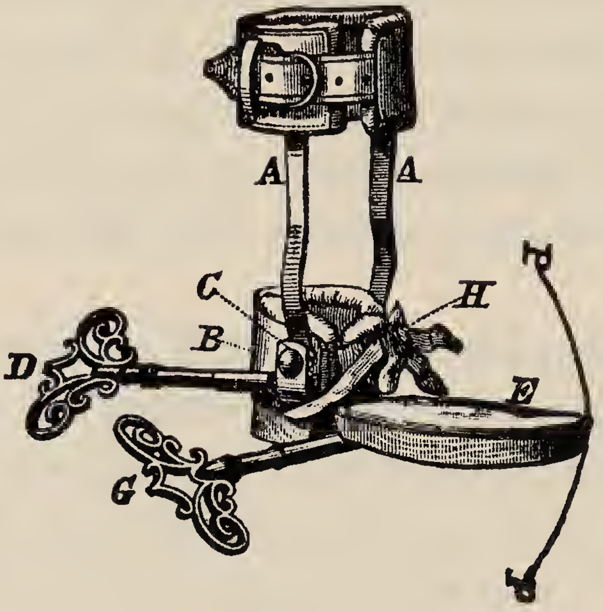

FIG. 9

The apparatus as at present used, without the webbing heel-traction strap (by which traction is made), is shown in Fig. 10. It consists of a calf band joined to two strong uprights, which are attached, opposite or below the center of ankle-joint motion, to the heel-cup. These parts, being very prominent and easily understood, have not been lettered. This heel-cup[1] is perforated posteriorly, as in the conventional form of apparatus, by two openings, H, through which the heel-strap, or, as I prefer to call it, the astragalus retention-strap, is passed (see C, Fig. 16). One upright (see Fig. 12, C D) is attached to the heel-cup by an endless worm and screw, controlled by the key A (Fig. 10); the other upright has a plain antero-posterior joint, F, seen in the engraving. This endless worm and screw movement enables the operator to place the foot-plate of

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[1] This heel-cup is an unnecessary adjunct to any club-foot shoe which aims to bring the heel downward and forward. I have recently had my apparatus made without any heel-cup. It serves, however, as a useful guide in placing the heel in position before applying traction, and strengthens the apparatus. But the Scarpa heel-cup is unnecessary, inasmuch as the first movement of the heel is *forward* and *downward*.

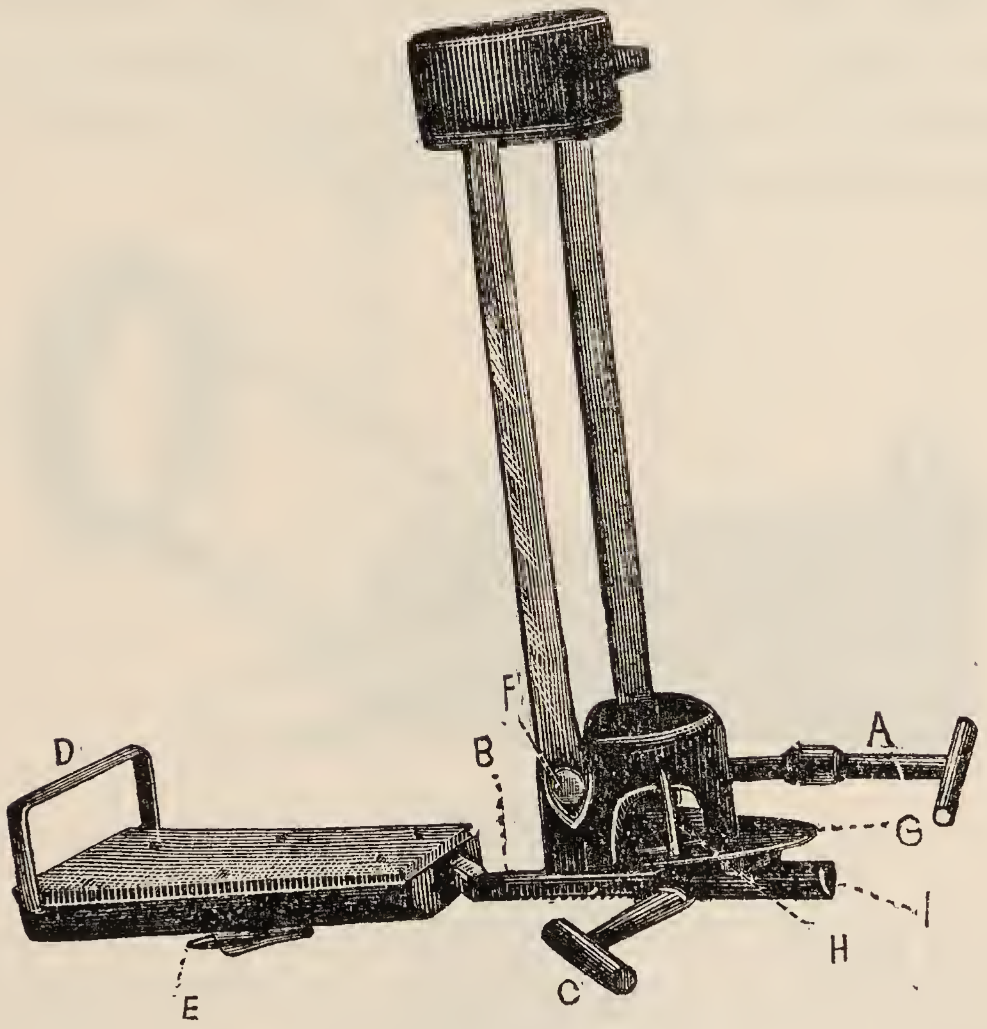

FIG. 10

the apparatus at any desired angle of flexion or extension. At a point which corresponds, when the apparatus is applied, with the medio-tarsal joint, the foot-plate is divided transversely and the anterior part is connected with the heel portion by a traction rod, B, which is controlled by a key C. Under the heel-cup is a long cylinder, I, into which the traction bar fits, and the foot-plate is extended posteriorly to afford a strong support (G) for the traction-cylinder and rod. Anteriorly the foot-plate has on its lower surface two buckles, E, for the attachment of the traction heel-strap (Fig. 14; Fig. 15; K and D D, Fig. 16). This heel traction-strap is made of webbing. It goes around the heel, and, passing forward, is reflected back upon the uprights of the bridge, D, to the buckles at E, on either side of the under part of the foot-plate (see Figs. 15 and 16). The key C moves the anterior part of the foot-plate backward and forward by a ratchet movement on the extension bar, B.

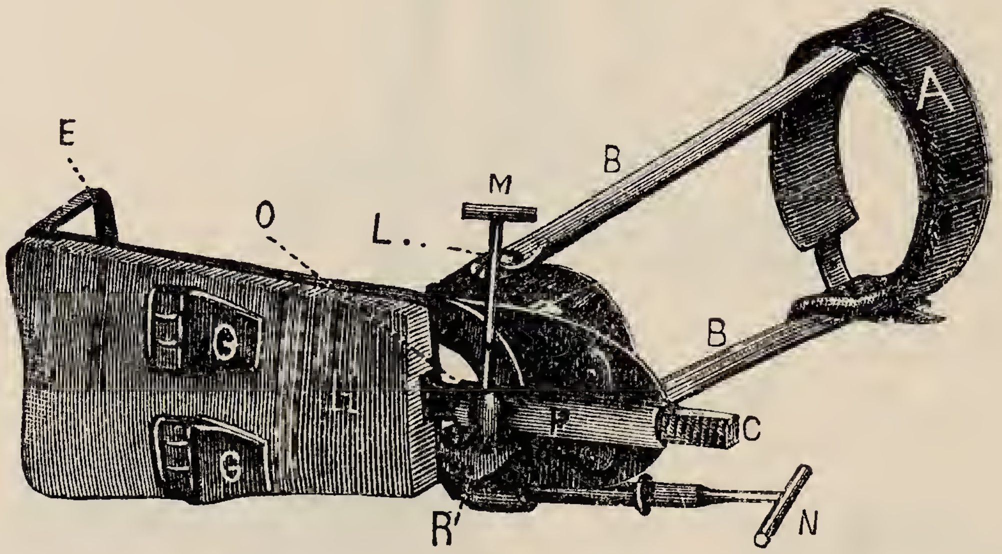

Fig. 11

Fig. 11 gives a view of the lower part of the foot-plate. The buckles, G G, for the attachment of the traction heel-strap; the traction rod, C; the cylinder, P; the flexion and extension worm and screw controlled by the key, N, are plainly shown. The key, M, is in position to throw the anterior part of the foot-piece forward. The semicircular opening, O, for the descent of the heel, in the foot part of the heel-cup, surrounded by its strong rim, R, is also shown. When traction is made and the heel is brought downward and forward, it passes through this opening unopposed by any obstruction.

Fig. 12 shows another view of the apparatus. The endless screw is shown at C and D; the heel-cup at E, with the two openings, M M, for the astragalus retention-strap; the semicircular opening, O, for the descending heel, and the bridge, F, for the reflected heel traction-strap.

Fig. 13 affords a general view of the apparatus with the traction rod closed and with the heel, toe, and astragalar arrows in position, all having their center in the ankle joint, C, of the apparatus.

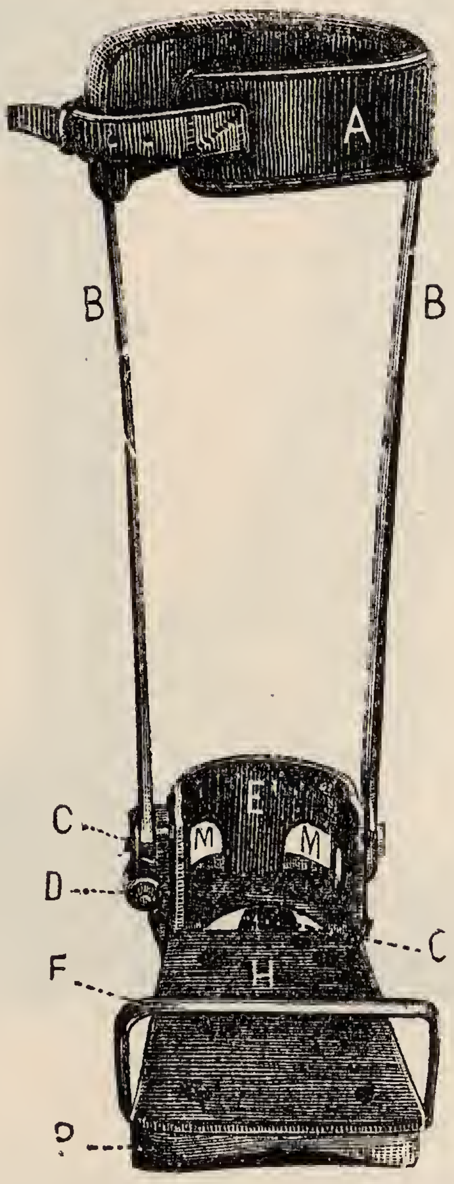

FIG. 12

This being a brief description of the antero-posterior traction apparatus, I will now attempt to show its application and to demonstrate how it fulfills the indications I have tried to point out.

The first step in the application of the apparatus is to prepare the foot for the traction it must undergo, and to so apply the necessary pads and straps that the movements indicated in the previous part of my remarks may be accomplished.

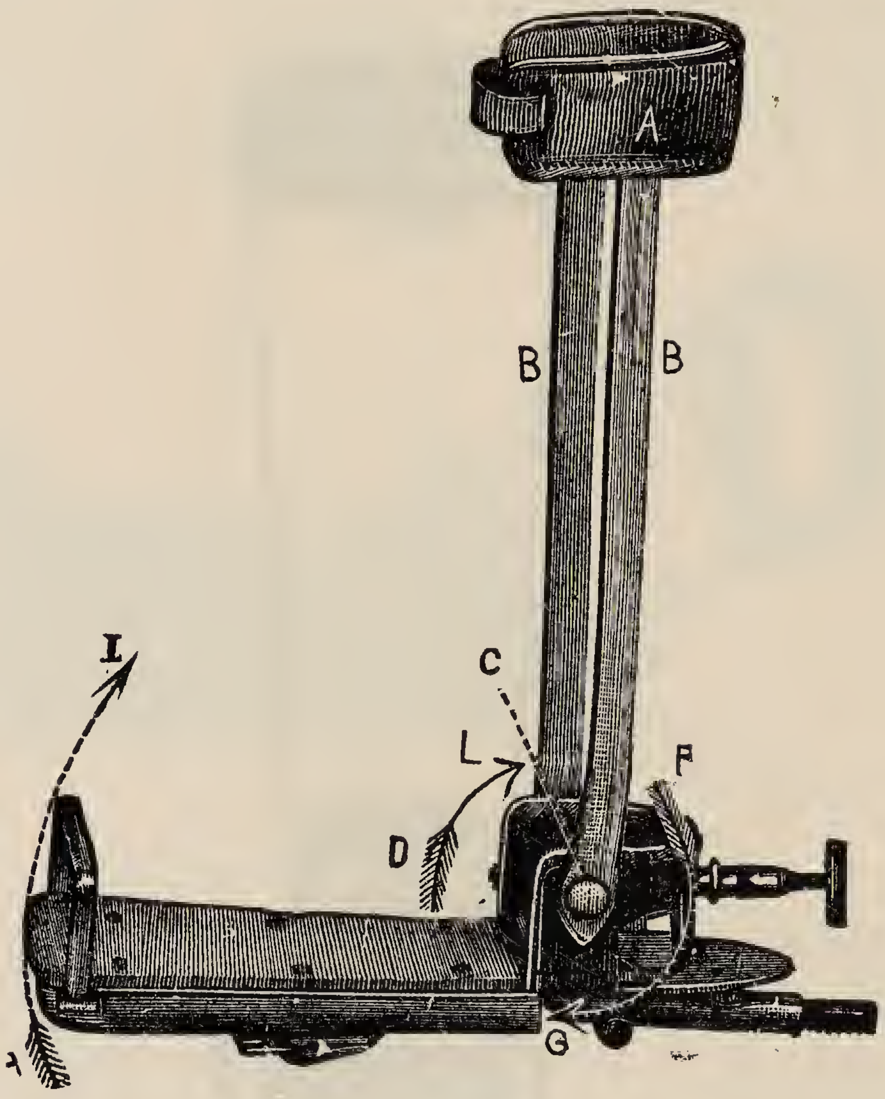

FIG. 13

This preliminary process is shown in Fig. 14. A thick pad of Canton flannel or other soft material, C, is placed over the neck of the astragalus. A narrow piece of roller bandage, A, is then passed over it and around the heel as indicated in the engraving. Two or three turns are sufficient. The end of the bandage is secured by an ordinary pin just below the internal malleolus, where no pressure is exerted when the traction is applied. A piece of surgical webbing five eights of an inch wide is then placed over the heel, well down, and brought forward under the bandage and below the malleoli (as shown in Fig. 14), the bandage acting not only to retain the astragalar pad in position, but the heel traction-strap as well. (In the engraving the webbing heel traction-strap is much too short and the pad, C, should be much thicker.)

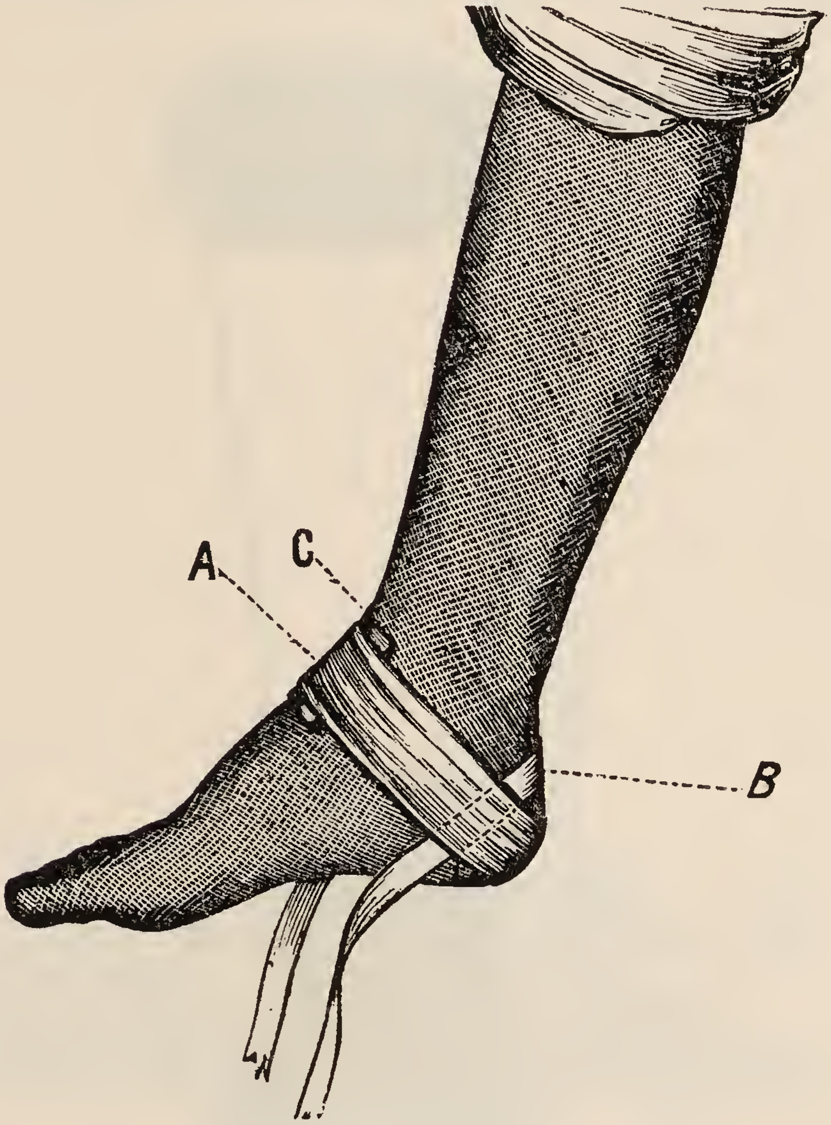

Fig. 14

In Fig. 15 the traction heel-strap is shown (D D and K). This is the correct position for it when the traction is applied, though it is here shown lying against the heel-cup at K, without being passed under the retaining bandage, as in Fig. 14. It is carried forward to the uprights of the bridge and is reflected back at O, to pass, as in J (Fig. 16), to the buckles underneath the foot-plate.

In Fig. 16 the astragalus and heel traction-straps are shown in position, the general adjustment being illustrated in Figs. 17, 18, and 19. The astragalus-strap, C, is

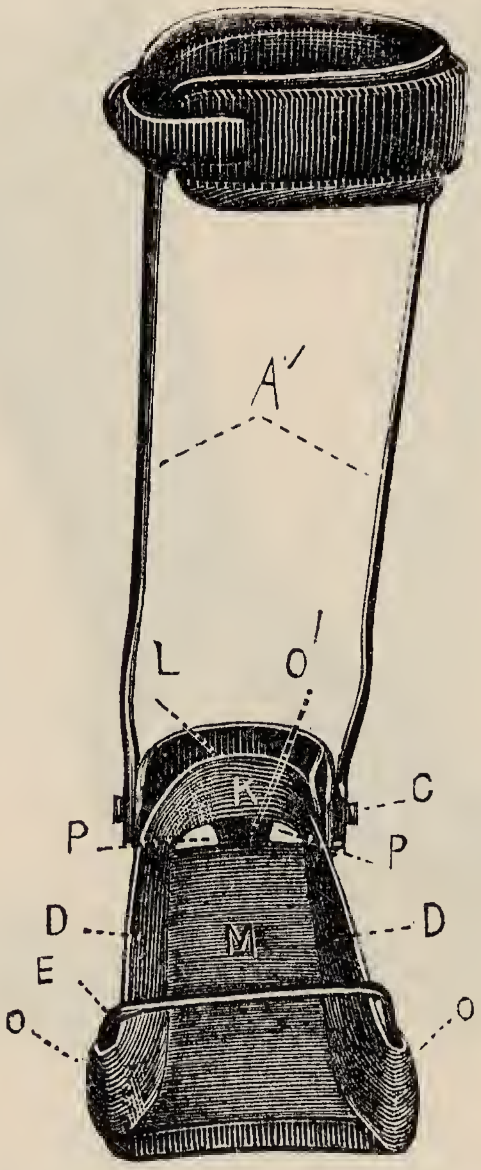

Fig. 15

made of Allien's webbing, five eighths of an inch wide, seventeen or eighteen inches long, and a buckle is sewed on one of its free ends, as at D (Fig. 16). This webbing is passed through the openings in the upright part of the heel-cup (M M, Fig. 12), and is "buckled on itself," as shown in the engraving. The traction heel-strap is also shown (H J), though, of course, this strap is passed around the heel and under the retaining bandage as a preliminary step, as shown in Fig. 14.

I have made it a cardinal rule in the treatment of chronic deformities *to make the apparatus fit the deformity*, and to so arrange the mechanism of the apparatus that a progressive and easily applied pressure may be exerted in the direction indicated. This is especially true of the mechanical treatment of club-foot, and I have long since ceased to attempt to crowd a crooked foot, partly relieved by tenotomy, into, comparatively speaking, a straight apparatus.

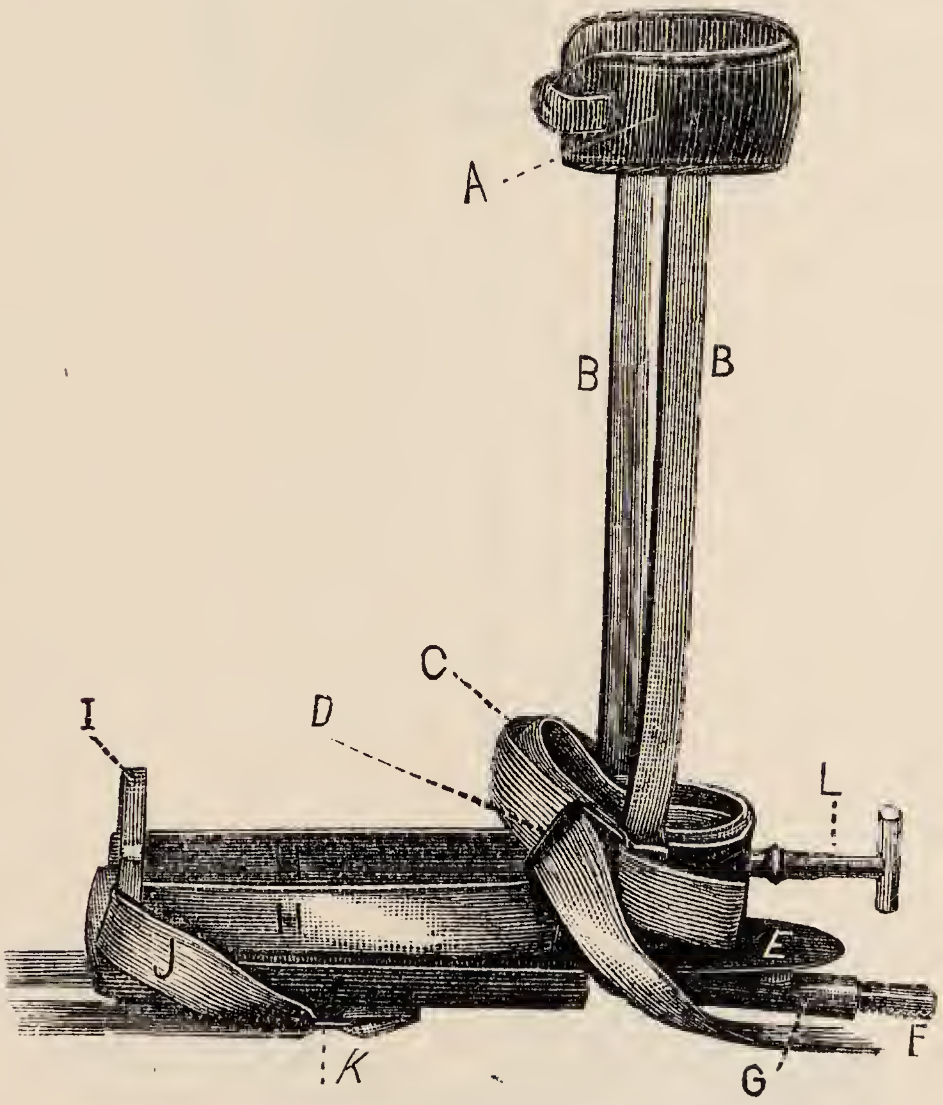

Fig. 16

Adapting the antero-posterior traction apparatus exactly to the antero-posterior deformity by using key L, Fig. 16, and placing the foot, with its traction heel-strap and astragalar pad adjusted as in Fig. 14, in the apparatus,

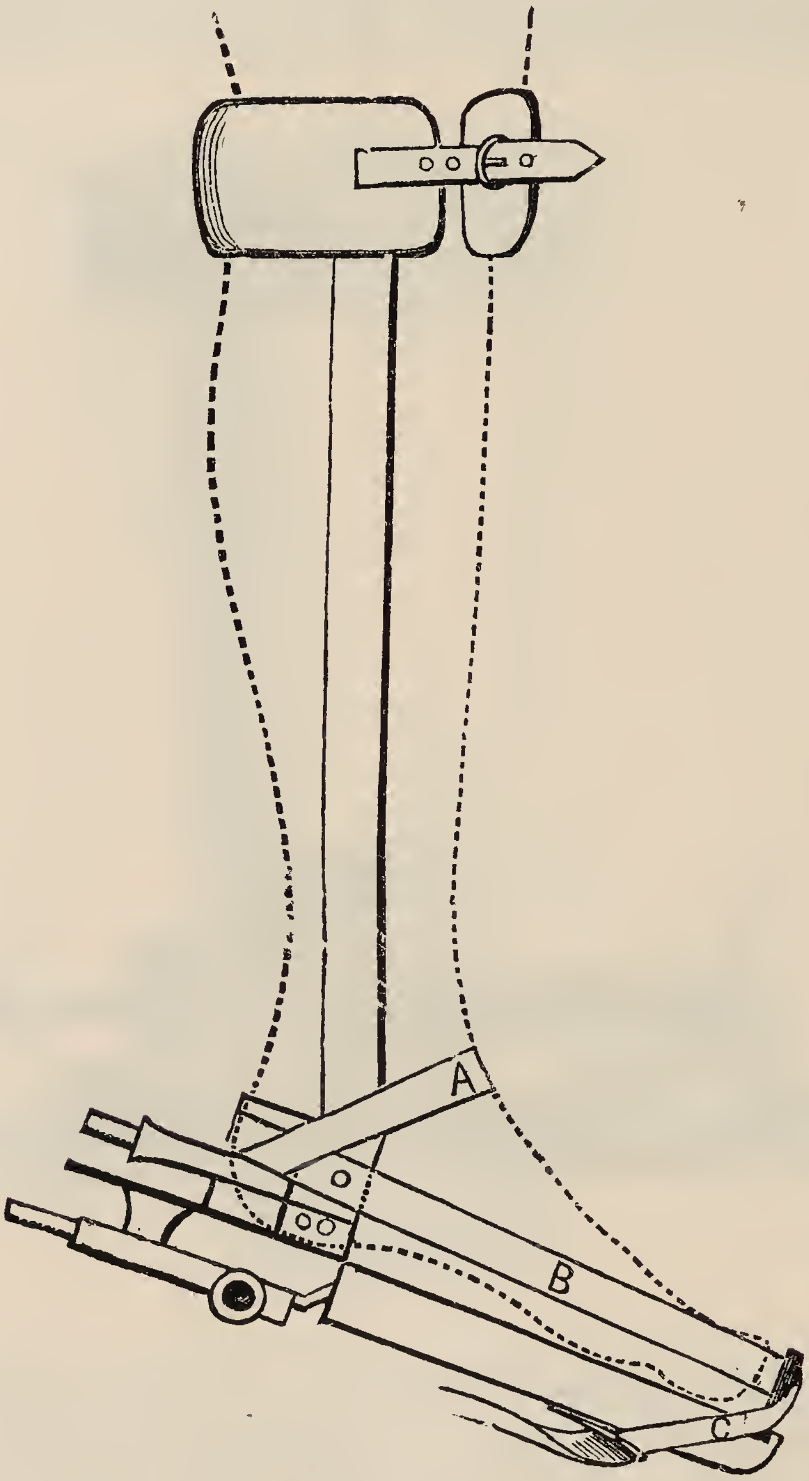

Fig. 17

we have an adjustment as shown in Fig. 17. The astragalar strap, A, is buckled on itself, holding the foot securely in the heel-cup; the traction heel-strap, B C, is drawn tightly, reflected back around the vertical uprights of the "bridge," and buckled, each one of these webbing straps being made as tense as possible. The apparatus fits the

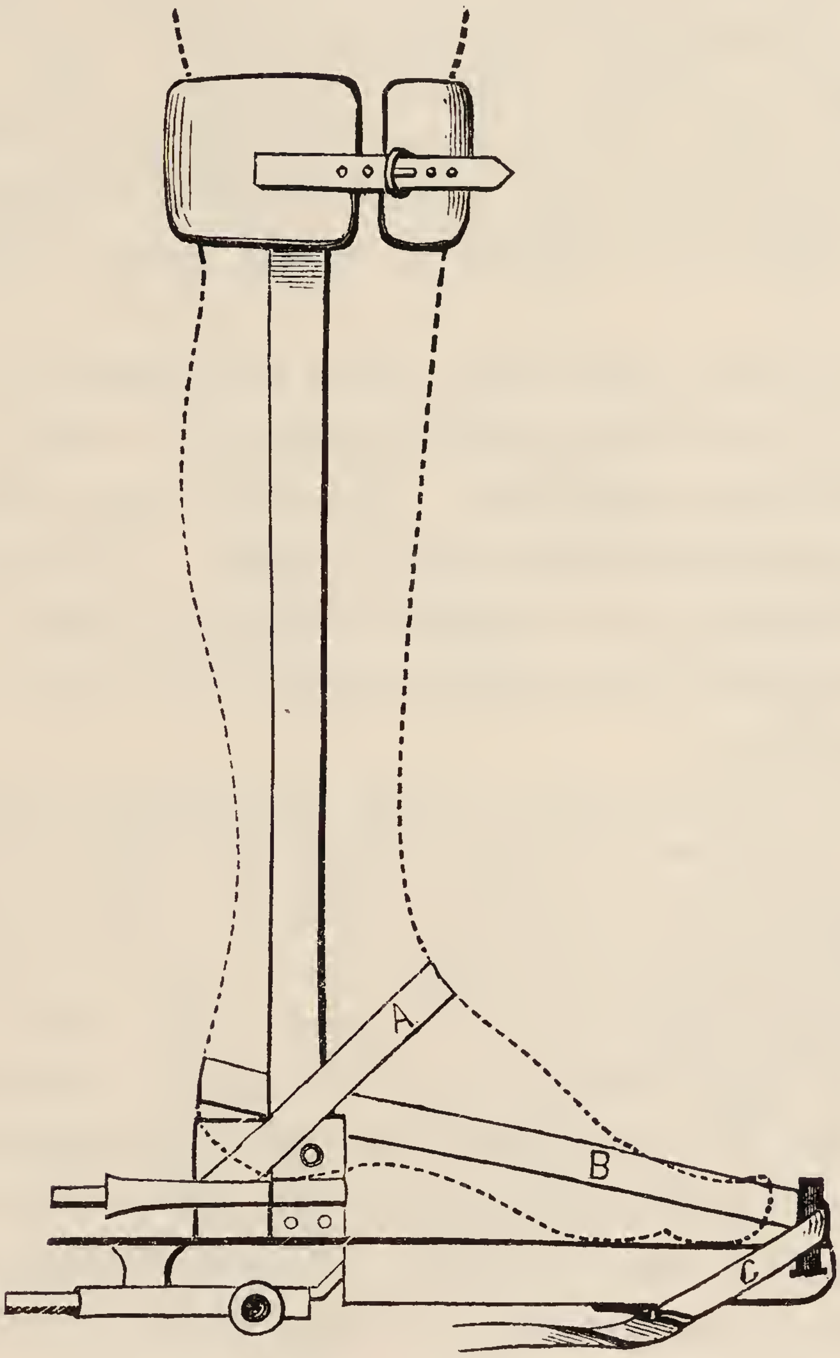

Fig. 18

deformity and the heel is squarely in its place, being retained by the astragalus strap. The astragalus strap, A, is made very tight in order to get an accurate and tense adjustment of the traction heel-strap, B C. Being sure that B C is drawn tightly, with the heel well down, and resting against the posterior part of the heel-cup, we now *loosen the astragalar strap*, A, *from half an inch to an inch and a half*, depending on the size of the foot. All being ready, we now use the key A (Fig. 10), and change the position of the apparatus from extension to that of the right-angled position. What happens? The apparatus obeys, but the foot does not change its position; the loosened astragalus strap allows the heel to take its own position, and no traction is made upon the resisting tissues.

Fig. 18 shows this result. The foot-plate of the apparatus is at the right-angled position, but the post-tibial contractions are unaffected. There has been nothing done to bring the heel *downward* and *forward*. On the contrary, it has remained in its original position, because the force thus far used has been applied upon the incorrect principle shown in Fig. 6.

We now give the key A (Fig. 10) another turn or two and use the antero-posterior worm and screw to force the apparatus into the position of flexion. The heel still remains in its original position. Now, however, use key C (Fig. 10) and apply direct traction. The result is shown in Fig. 19. The heel is drawn irresistibly *downward* and *forward*, the toes are carried *upward* and *forward*, and the *loosened astragalus strap permits the neck of the astragalus to move upward and backward*. By this simple process we have given the apparatus the desired plane of flexion, have made full allowance for the rotating astragalar neck, and have deliberately drawn the resisting foot into the position sought for, and fulfilled the indications presented in Figs. 4 and 5.

It will be noted not only that the normal antero-posterior movements of the ankle-joint are here reproduced, but that they are *exaggerated*. We can, by the use of this apparatus, imitate the natural movements, and, by the use of traction, stretch all the opposing antero-posterior tissues, whether post-tibial or plantar, or both. It not infrequently happens that the adolescent foot is permanently elongated half an inch by this easily controlled traction-force without tenotomy, and a temporary gain

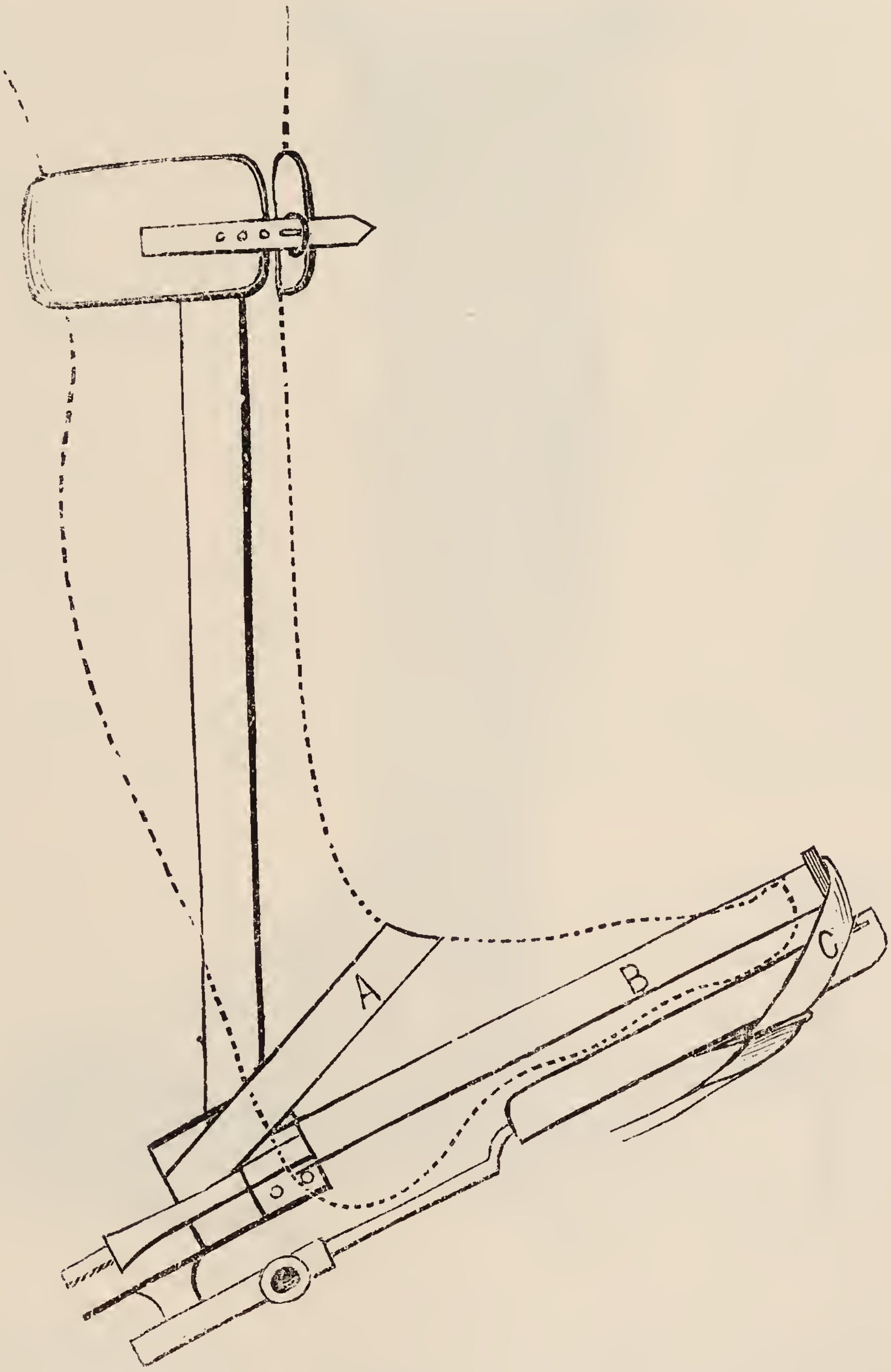

Fig. 19

of from one eighth to one fourth of an inch at a single treatment, lasting only fifteen or twenty minutes, is not unusual. This makes this apparatus particularly serviceable in adult or adolescent cases of non-deforming club-foot,[1] as well as in confirmed talipes equinus.

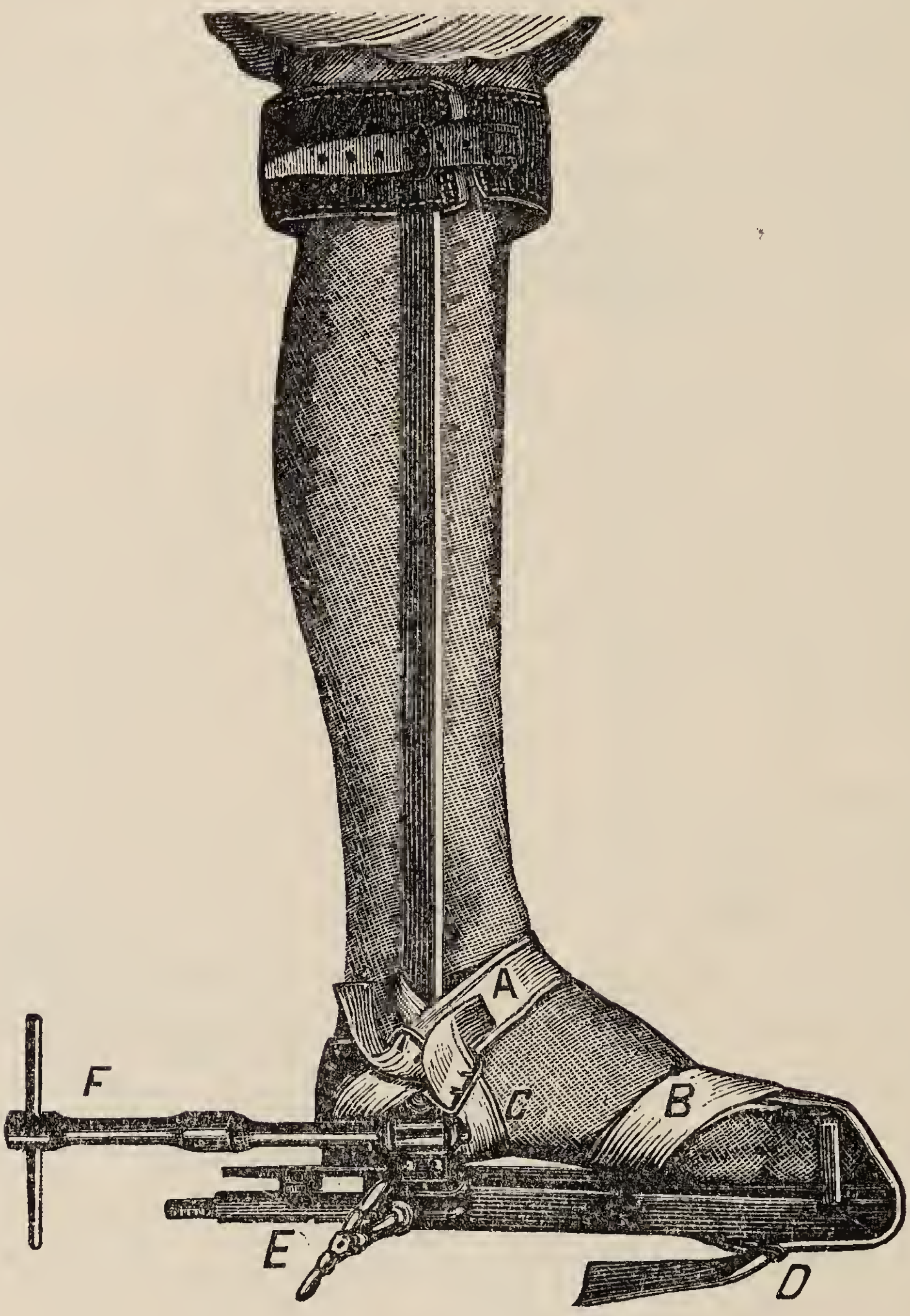

FIG. 20

We can make this traction apparatus available also in the, strictly speaking, tarsal or plantar contractions, especially when they are antero-posterior. When much varus exists, the "lateral-traction shoe" is indicated, and this will be described further on.

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[1] See "Non-deforming Club-Foot," by the writer, *Medical Record*, May 23, 1885.

In Fig. 20 the antero-posterior traction-shoe is applied to a patient with marked contraction of the plantar fascia. The astragalar strap is fastened in the manner described at Fig. 16. In place of the traction heel-strap there described, however, a webbing strap (C, Fig. 20) is passed underneath the os calcis and behind the lateral uprights of the apparatus, "buckling on itself," as does the astragalus strap (A, Fig. 20). We then have the os calcis secured firmly in the heel-cup by the astragalar and sub-os-calcis straps. A loop of webbing is then passed over the anterior part of the foot, just back of the "ball of the foot" (B, Fig. 20), and this is fastened to a buckle on the under part of the foot-plate (D, Fig. 20). The apparatus is now placed in any desired position of flexion, and direct traction is applied with key E, Fig. 20, and the result is shown in Fig. 21.

The more firmly the astragalus and the os calcis are fixed, the better in this condition, as the traction is needed at the plantar fascia, and not at the gastrocnemius muscle, and hence the astragalus strap is not loosened, as in the treatment of post-tibial contractions. I have seen severe and apparently permanent contractures in the adult overcome without tenotomy by this simple method of treatment, and it is apparently an easy matter to lengthen the foot from one eighth to one half of an inch in adults and adolescents by the traction method in cases of plantar contracture.[1]

Let us now briefly study the conditions found in equino-varus, and attempt to demonstrate some of the mechanical points involved, before describing the "lateral-traction shoe."

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[1] In Figs. 20 and 21 the necessary pads over the astragalus and under the metatarsal loop are omitted in order to show the principle of action more clearly. No pad is needed under the os-calcis strap.

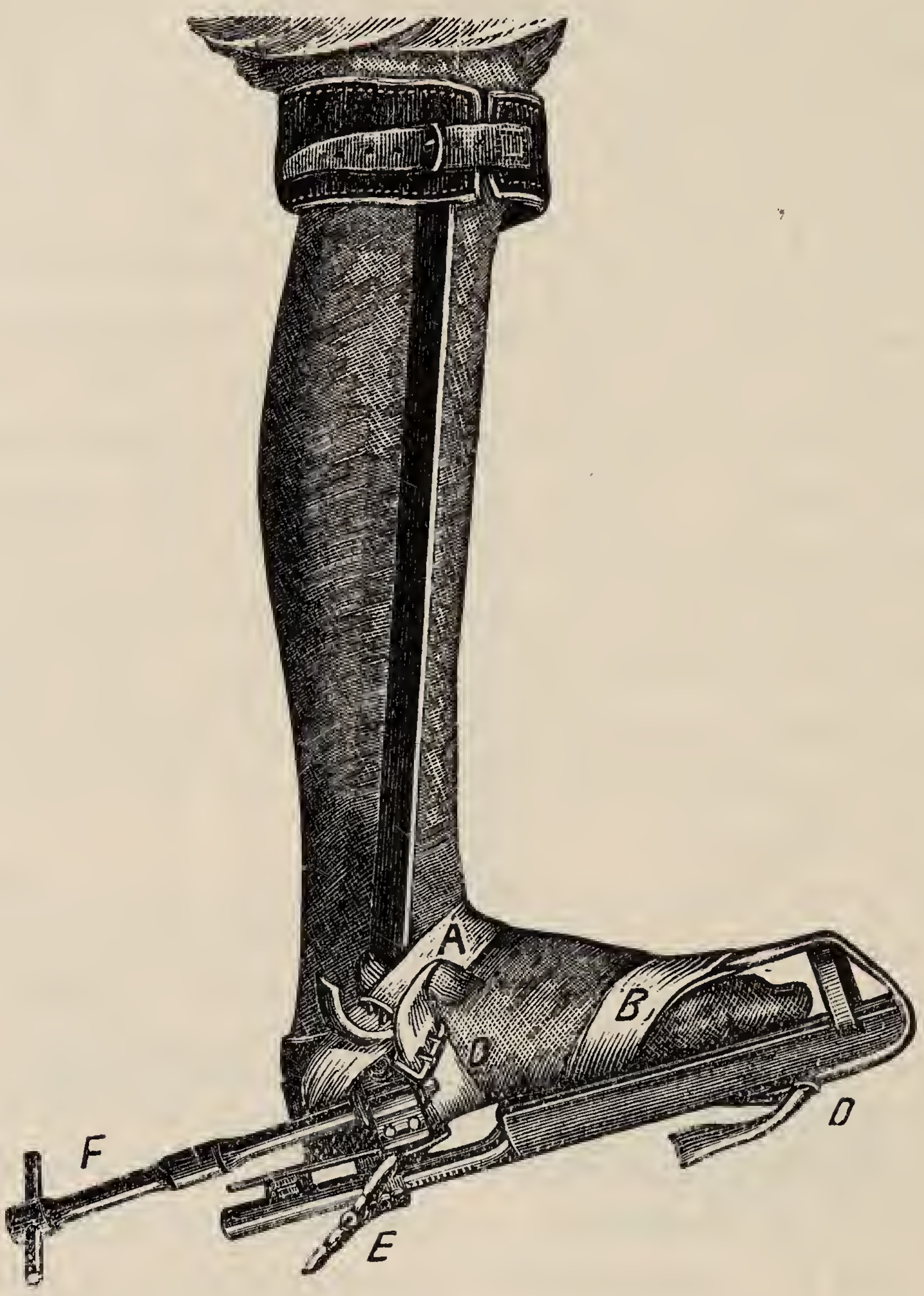

Fig. 21

As before stated, I formerly used the simple "lateral pusher" in treating the lateral malpositions of equinovarus, and I reproduce the original engraving that was used in my first paper on "Traction." It is shown in Fig. 22. Applied to the inner side of the foot and leg, it was intended to overcome the lateral deformities of equino-varus by a *pushing* force. It is, so far as I know, the only apparatus based upon the, strictly speaking, *pushing* principle (acting from a point above the tarsus) introduced for the treatment of the lateral deformities of talipes, though I inadvertently described it in my first essay as a "modification of Taylor's ankle support." In the *Hospital Gazette and Archives of Clinical Surgery* for January 9,

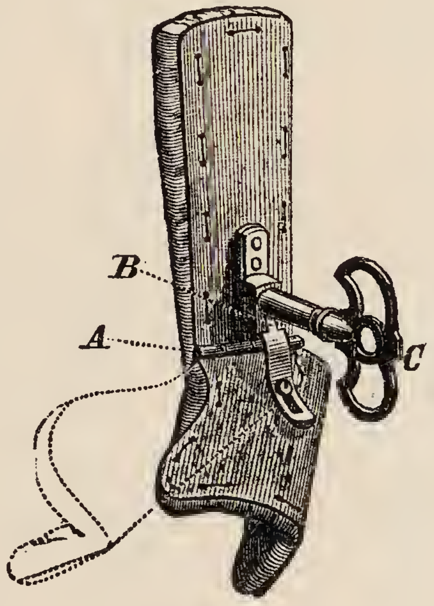

Fig. 22

1879, about seven weeks after my first article appeared, I called attention to my error, and claimed the apparatus as my own.

After a good deal of experience with this simple "pusher," I felt that it represented a correct principle of mechanical treatment, and that a lateral *pushing* force possessed many advantages over the lateral *pulling* force ordinarily employed. And, after many experiments, I have at last perfected a lateral *pushing* and *traction* apparatus, which enables the operator to apply a positive traction force to the deformity known as equino-varus.

But this term *equino-varus* is really a misnomer. There is some equinus present, for the heel is raised. There is some varus present, for the tarsus is turned in. If these conditions were all that marked the deformity known as equino-varus, the artificial mechanism devised for its relief need not be very comprehensive. But simple, uncomplicated equino-varus is rare in my experience.

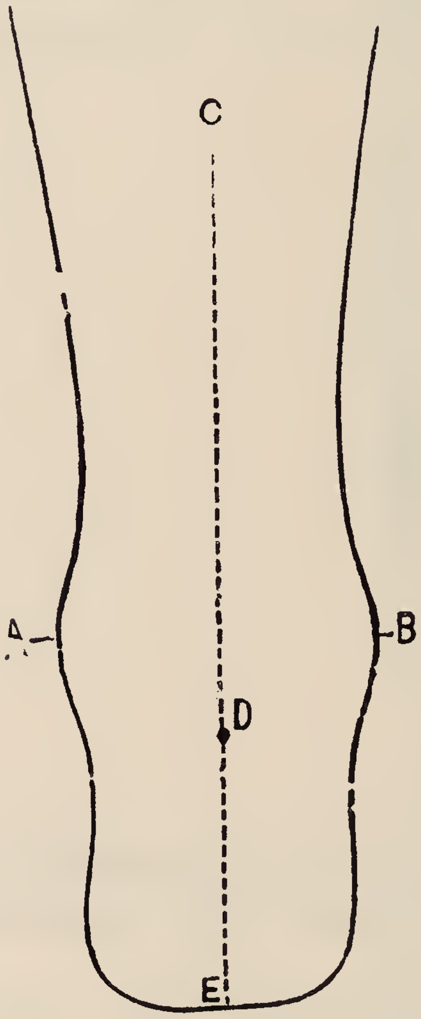

FIG. 23

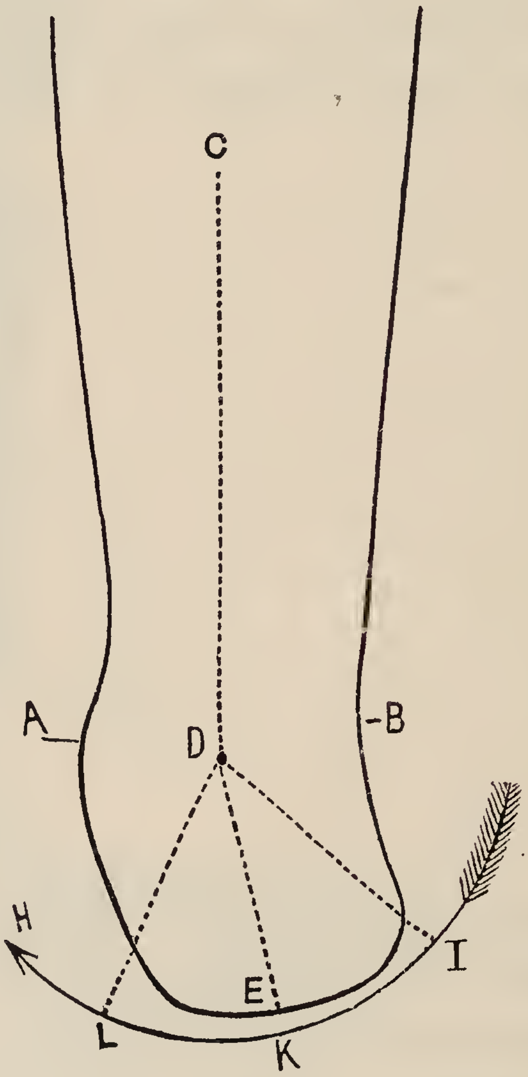

FIG. 24

Fig. 23 represents a sectional outline (reduced) of the normal (right) ankle of a boy of eight years. A represents the outer malleolus, B the inner malleolus. The line C D E passes through the ankle-joint to the heel, E D representing, in a diagrammatic sense, the center of the antero-posterior rotation.

In Fig. 24 we have the same sectional view of the right ankle of a child of the same age with an ordinary equinovarus.

The outer malleolus, A, is very prominent and apparently thrown backward; the inner malleolus is not perceptible, and can not be found at the point B.

When found by palpation, the inner malleolus is discovered to be apparently in front of its normal position. The straight line C D E, of Fig. 23, changes its direction at D, in Fig. 24, and it is deflected toward the mesial plane of the body. In short, the os calcis is not only drawn up by gastrocnemius contraction, but is rotated inward on an antero-posterior axis. This is a marked feature of the deformity.

But this is not all, nor is it the worst feature of equino-varus. The anterior part of the tarsus, and to a considerable extent the astragalus, are rotated inward on a vertical axis, thus accounting for the apparent loss of relation between the two malleoli. The malleoli are normal, or nearly so, but the astragalus is turned in, and much of the deformity is due to this rotation of the astragalus.[1]

This can be better understood by showing with illustrations the location of the malleoli in equino-varus.

Fig. 25 represents the normal outline (reduced) of the right foot of a boy of eight years old. The relation of the two malleoli to the sole of the foot is shown at A (the outer) and B (the inner). The line C D E passes through the long diameter of the sole of the foot from "heel to toe," through the point located diagrammatically at D.

Fig. 26 is taken from the right foot of a boy, aged eight years, with equino-varus. The outer malleolus is at A. The inner is at the point corresponding with B, and the straight line C D E, in Fig. 25, becomes a crooked line, the part anterior to the point D (D E) forming an obtuse angle with that which passes from D to C.

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[1] I am aware of the fact that rotation of the astragalus has been pointed out by several writers on club-foot. But I feel that sufficient attention has not been paid to the important part it plays in the production of the deformity, or to the obstacles which this rotation presents in treatment.

From a standpoint of mechanical treatment, the indications, so far as rotation of the tarsus, and especially the astragalus, upon a vertical axis is concerned, are plainly shown by combining these two diagrams in one. And this has been done in Fig. 27. Using the heel as a fixed point, the effort should be made to throw the anterior part of the foot in the direction of the curved arrow, G H, until the normal position, or an exaggeration of the normal position, is reached. When this is done, the two malleoli will be found in their proper positions as shown in the diagram (Fig. 27); the rotation of the astragalus and the anterior

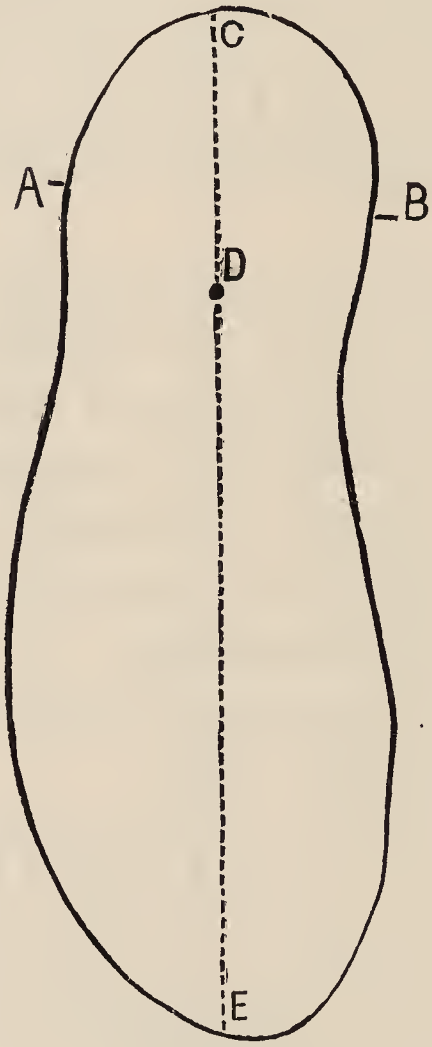

Fig. 25

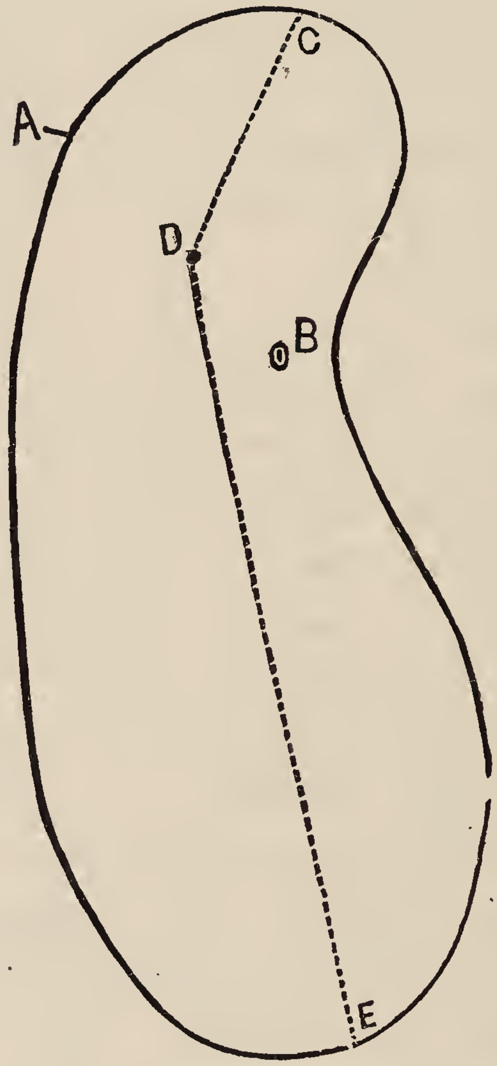

Fig. 26

part of the tarsus upon a vertical axis will be overcome, and the leg and thigh will be found to occupy a normal relation to the tarsus.

In equino-varus (I speak especially of the congenital type) there is ordinarily no deformity or perceptible deviation from the normal in the tibia or fibula. The real deformity occurs at and below the ankle-joint, the rotation of the astragalus inward, upon a vertical axis (with the ankle in extension), making an apparent outward rotation of the leg and thigh.[1] Under these circumstances, if the leg and thigh are placed in normal position, the malleoli

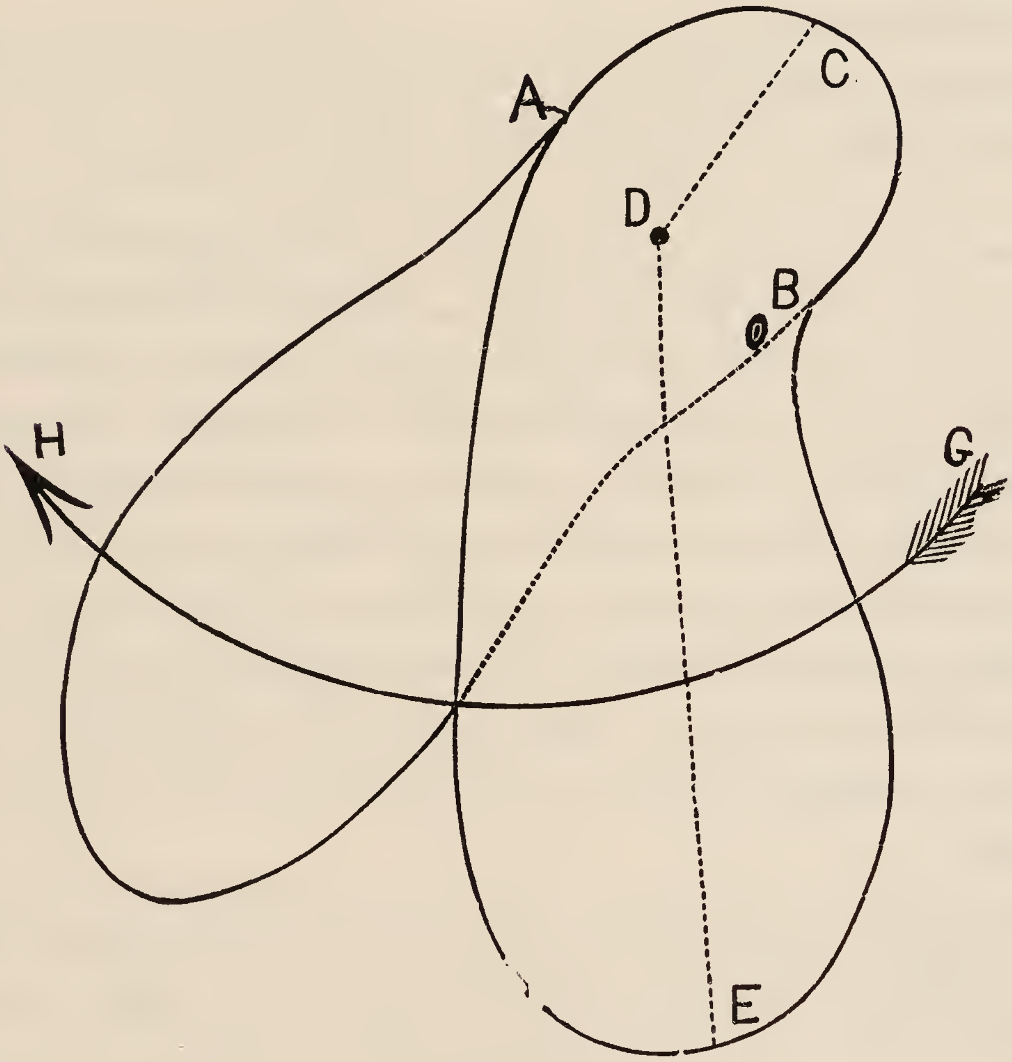

FIG. 27

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[1] Rotation of the leg and thigh, with the knee in extension, has its seat at the hip-joint.

are found in their proper positions. It will then be readily appreciated that the deformity occurs at a point below the inferior articulating surface of the tibia. The really difficult "lateral" deformity is not at the medio-tarsal joint, as so many teach, but is found in the rotation upon an antero-posterior and a vertical axis of all the important bones of the tarsus.

Comparing, again, Figs. 25 and 26, it will be found that the inner border of the foot is greatly curved. It is also raised, so that the cuboid bone presents in walking. This is due to the rotation upon both an antero-posterior and a vertical axis of the whole tarsus. In brief, there are three principal deformities in equino-varus: First, rotation of the ankle upon a transverse axis (extension); secondly, rotation of the os calcis upon an antero-posterior axis (inversion of the os calcis); and, thirdly, rotation of the tarsus upon both an antero-posterior and a vertical axis, including rotation inward of the astragalus and anterior portion of the tarsus. These three deformities are each met by a specially devised movement in the lateral-traction shoe.

As in lateral curvature, it is not the simple lateral deformity that is so difficult to remove as the rotation of the vertebral bones which emphasizes it, so in equino-varus it is not the simple lateral and uncomplicated antero-posterior displacements that present the greatest difficulties. It is the compound rotation of the tarsus, and especially the rotation of the astragalus and os calcis, that makes the problem of relief so difficult.[1]

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[1] It is impossible for any one to voluntarily place the foot, by muscular action, in the position of confirmed equino-varus, with rotation of the astragalus; just as it is impossible for any one to place the vertebral column in the position of confirmed lateral curvature with rotation by voluntary muscular action. These facts have, it seems to me, a peculiar significance, and bear directly upon the question of ætiology. They are both exaggerated

The first indication, from a mechanical standpoint, in the treatment of equino-varus is to overcome the inversion of the os calcis (see Fig. 24) so that a fixed point may be secured upon which to apply a rotating traction force in the direction indicated in Fig. 27. The curved arrow, I K L H, of Fig. 24, shows how this force should be applied, and a glance is sufficient to show that a *pushing* force, purely lateral, is indicated. The hinged lever and screw of the simple lateral pusher (Fig. 22) meets this indication perfectly.

The second indication is to find a center of motion (the first lateral push having been applied to the os calcis) which will permit the application of a traction force that will remove the rotation of the astragalus and carry the anterior portion of the tarsus with it. The direction of this force should be *forward* and *outward*, and a study of Fig. 28 will demonstrate that this center is at a point located at or outside of the outer border of the foot in the region of the articulation of the os calcis with the cuboid bone. This point is indicated at the letter C (Fig. 28), and the curved arrow, I P E F G H J, gives the direction required, viz., forward and outward. This is the second indication.

But the heel is raised, and, unless this malposition can be removed, the lateral push to the os calcis and the rotat-

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positions, not induced by simple malposition, or simple muscular action, or both. Both of these conditions present their worst features in the rotation of the small bones, which possess very limited movements in the direction of their exaggerated, deformed position.

It seems to me that both these and other allied conditions are due primarily to unilateral muscular action of the nature of a contracture or a localized arrest of muscular development, and that the exaggerated position of these small bones, their rotation, the contracture of ligaments, fasciæ, skin, and all the subcutaneous tissues, are the result of *rapid growth* in the acquired position. In no other way can I explain the exaggerated rotation, and the other phenomena observed.

ing (traction) force applied to the tarsus will amount to little or nothing.

As demonstrated in the first part of this paper, the heel should pass *downward* and *forward*. By attaching the heel traction strap to the traction rod which carries the anterior part of the apparatus *forward* and *outward* upon the center C (Fig. 28), this movement may be readily accomplished. This is the third indication.

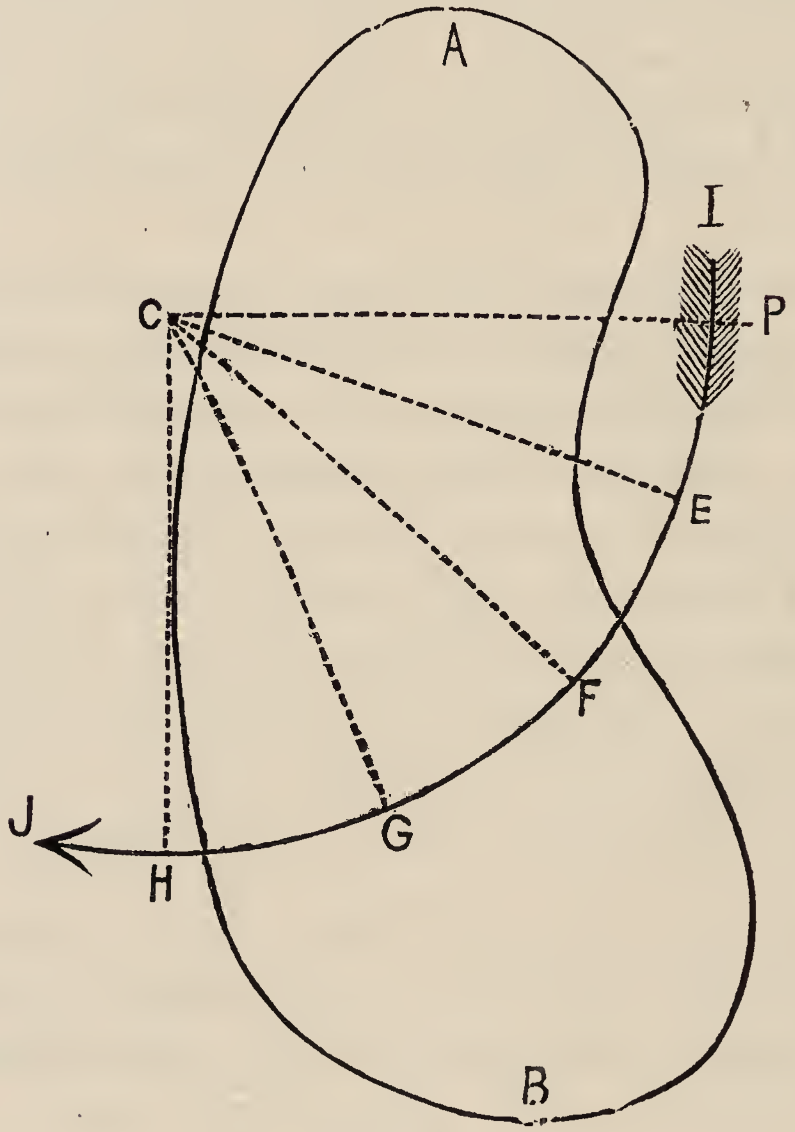

Fig. 28

While it is necessary to restrain the astragalus by an astragalar strap, the same as in the antero-posterior traction-shoe, in order to execute all these movements—viz., the lateral push, the rotating push, and the direct traction—the movement of the apparatus when applied to the conditions of equino-varus concentrates the counter-pressure at the dorsal portion of the articulation between the os calcis and the cuboid, and the acquired position of the apparatus, when all these three movements have been applied, represents the exact reverse of the deformity. In short, as in the antero-posterior traction-shoe, the apparatus being applied exactly to the deformity, the foot is forcibly drawn into a reverse position by means of a direct rotating traction, and all the resisting tissues are reached, including the ligaments and fasciæ as well as the muscles.

The lateral-traction apparatus is based upon these three indications (the three varieties of rotation mentioned above), and I will now describe it in detail.

It consists (Fig. 29) of a calf band, A, to which is attached a single upright of annealed steel, A^1. This upright is always placed on the side toward which the deformity looks; in varus on the inside, in valgus on the outside of the leg, the instrument being a *pusher*. At a point just above the center of motion of the ankle-joint is placed a *lateral joint*, C, which is acted upon by a hinged lever and screw; B^2, being a worm and screw, and O an arm which pushes the lower part of the apparatus toward the deformity to any desired extent. The distal end of this arm is free, and, as the arm is turned away from the deformity by key B^1, the foot part of the apparatus takes any lateral position required. All of the keys—B^1, E^1, and G^1—are removed, of course, after the desired lateral position is obtained and traction has been applied.

This lateral hinge, with its lever and screw, is intended to meet especially the inversion of the os calcis. It antagonizes, when traction is applied, the tibialis posticus muscle contraction as well as the shortening of the internal lateral ligament, which is frequently more troublesome than the muscular contraction. This first lateral push is important and essential.

Just below this hinged lever and screw is an antero-posterior joint, E, similar in all respects to that used in the antero-posterior traction apparatus described in the previous pages. It is a simple worm and screw controlled by the key E¹. Like the worm and screw in the antero-posterior apparatus, it enables the operator to secure any desired angle of flexion and extension, and to create a plane different from that of the deformity. The traction executed by this apparatus, like that in the apparatus first described, draws the foot into this acquired plane and stretches all the resisting tissues.

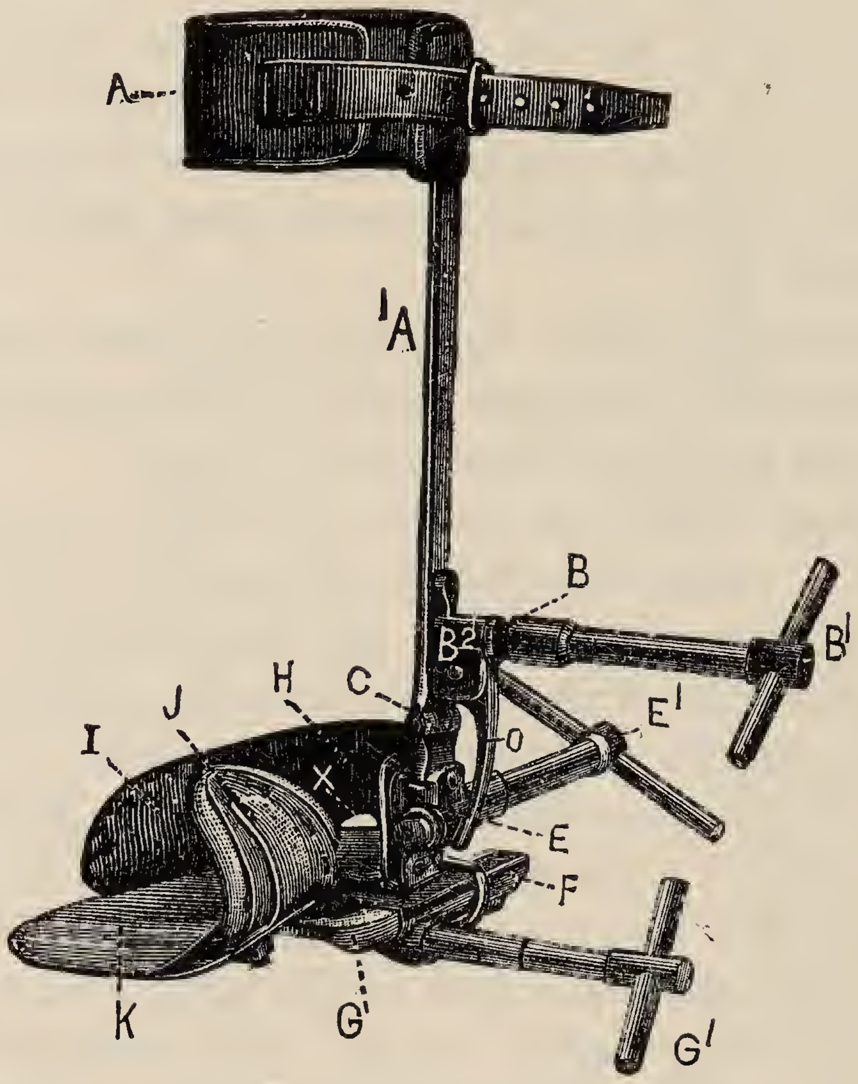

Fig. 29

Still lower and acting upon the anterior part of the foot-plate (which is divided transversely at a point corresponding with the medio-tarsal joint) is the traction rod placed upon the inner side of the apparatus and moving in the cylinder F. It is controlled by the key G^1. This will be more fully dwelt upon when we study the under surface of the foot-plate.

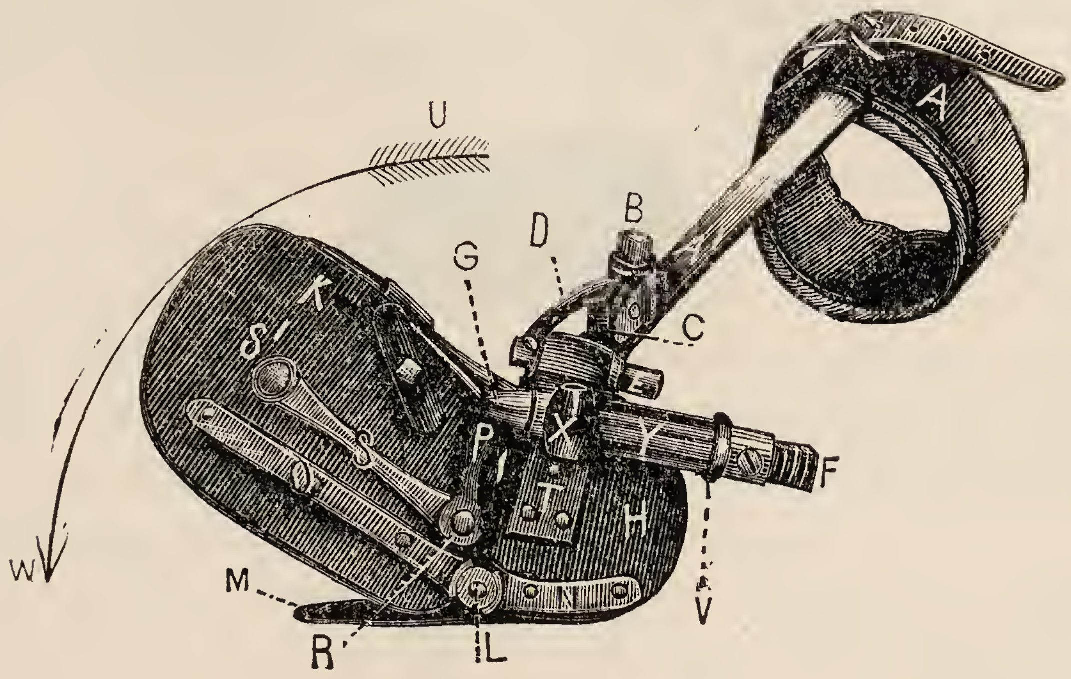

FIG. 30

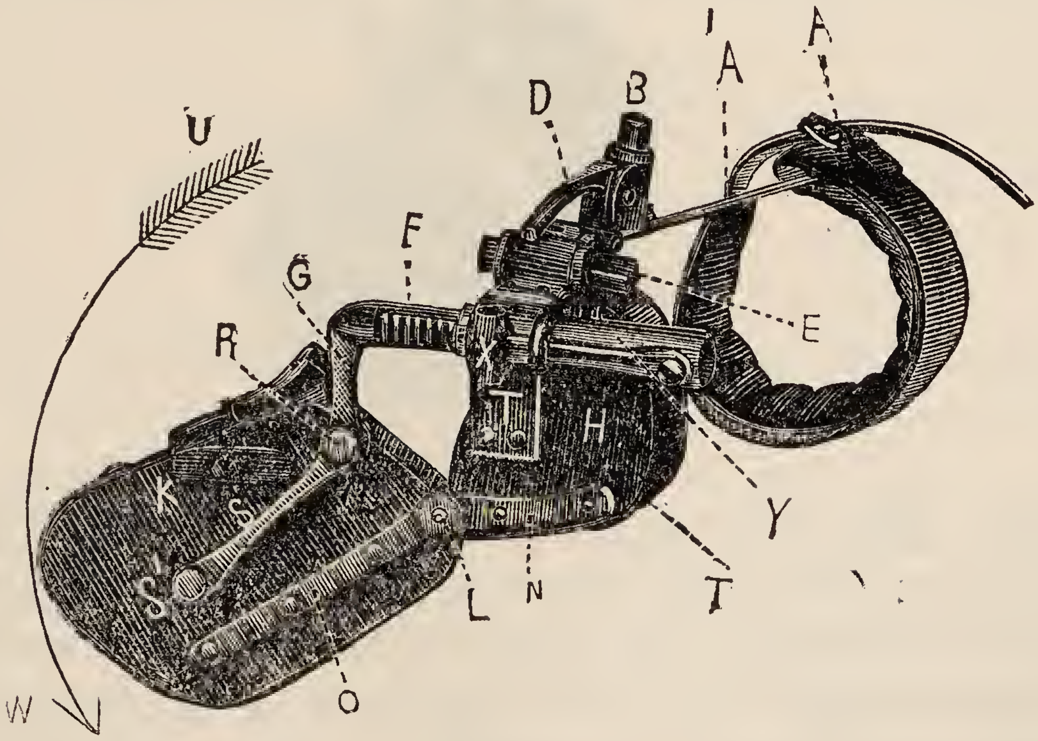

FIG. 31

There is the customary heel-cup (though this is not necessary any more than in the antero-posterior apparatus, X, Fig. 29, and H, Fig. 32) with the openings (K K, Fig. 32) through which the restraining astragalus strap is passed. The outer part of this heel-plate (M, Fig. 30; L, Fig. 32; and I, Fig. 33) is extended quite well forward at the outer border of the foot-piece to form a resistance to the rotating movement imparted to the foot by the traction rod G upon the center L. And the inner border of the foot-plate is curved over the dorsum of the foot (J, Fig. 32) in order to grasp it as it moves upon the center of motion at the outer border of the foot, as shown in

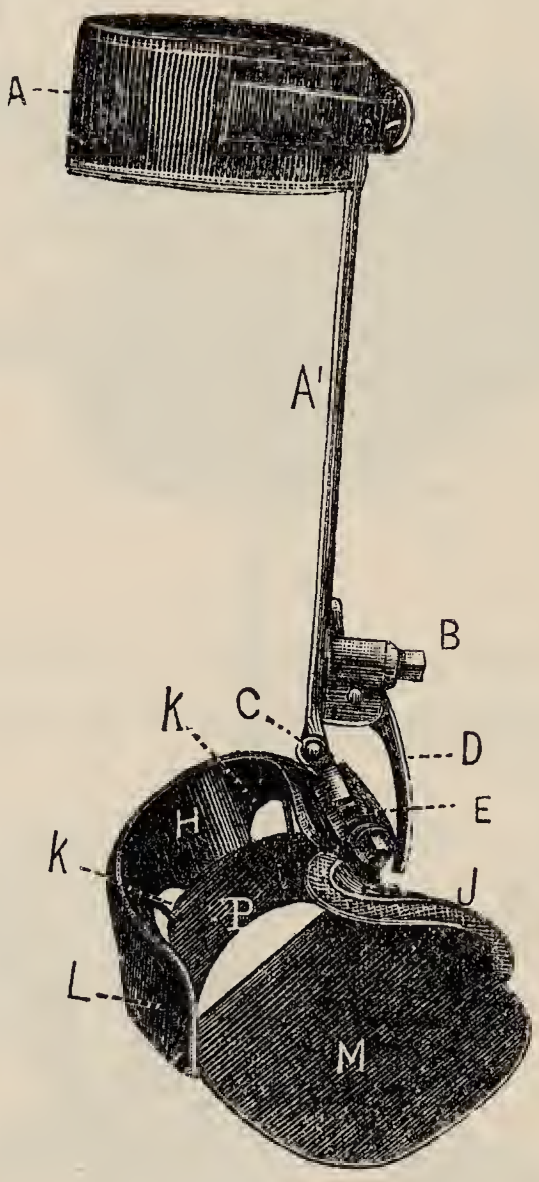

Fig. 32

Figs. 30 and 31, L. The heel-plate part of the heel-cup has the semicircular opening (P, Fig. 32) to permit the descent of the heel when the traction is applied, the same as in the antero-posterior shoe.

Figs. 30 and 31 show the lower surface of the foot-plate. In the former the traction rod is closed in the position of deformity; in the latter it is extended or thrown open, showing by the arrow, U W, the direction of the force applied, viz., *forward* and *outward* (compare with Fig. 28). At K, Figs. 30 and 31, is an arrangement by which the curved part of the foot-plate (J, Figs. 29 and 32) can be adapted to any position required.

Fig. 32 pictures the apparatus in a position of slight equino-varus. The lateral hinged lever and screw has been turned to meet the inversion of the heel. The antero-posterior worm and screw has been used to drop the foot into a position of equinus, and the traction rod is closed in a position of varus. By using the three movements described as being controlled by keys B^1, E^1, and G^1, in Fig. 29, a direct antagonizing traction force can be applied to almost any degree of deformity of the equino-varus type.

In Fig. 33 the astragalus strap, M, is shown. (The artist has made this webbing strap fully one half too wide.) It is passed through the openings in the heel-plate K K, Fig. 32, and is reflected upon itself and buckled.

The foot is prepared for this apparatus as pictured in Fig. 14. A soft pad of Canton flannel or other soft material is placed over the neck of the astragalus under a simple retaining bandage, and a traction heel-strap, with, however, a buckle attached to one end, is passed around the heel low down and the ends brought forward. The foot is now placed in the apparatus, which is made to exactly fit the deformity. The astragalar strap (M, Fig. 33) is then buckled tightly and the heel traction strap, not lettered in Fig. 14, is brought directly down, inside the heel-cup, and buckled underneath the foot-plate just anterior to the right-angled bar, P, of traction rod F (Fig. 30). This traction heel-strap is buckled tightly.

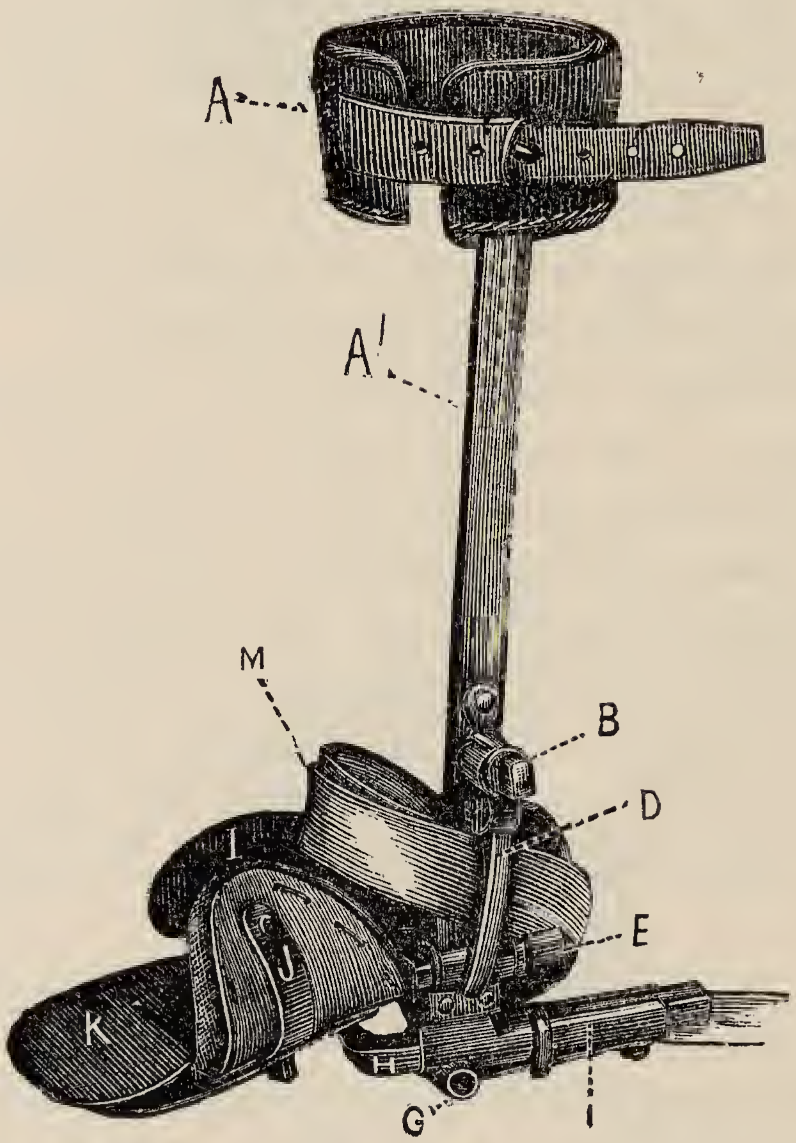

Fig. 33

(It seemed impossible to show this traction heel-strap in the engraving.) The astragalus strap (M, Fig. 33) is now loosened slightly to accommodate the upward movement of the astragalus when the ankle is forcibly flexed. All being in readiness, key B^1 (Fig. 29) is used to turn the hinged lever and screw which everts the os calcis. Key E^1 (Fig. 29) is then used to raise the foot-plate to the desired extent toward or above the right-angled position. When these two movements are satisfactorily executed, key G^1 is made to turn the foot-plate outward and forward upon the center (C, Fig. 28, and as in Figs. 30 and 31), into a valgus position, the traction heel-strap drawing the heel *downward* and *forward* as in the antero-posterior traction apparatus, while the anterior portion of the tarsus is rotated *outward.* All the movements indicated are accurately fulfilled. The traction heel-strap is especially necessary to carry the heel downward and forward, while the tarsus is being thrown forward and outward by the traction rod. If the heel is not controlled in this manner, the lateral rotating traction is rendered useless.

The location of the center of motion at the outer border of the foot-plate must be determined by the degree of lateral deformity. I have for years placed it at different points; at the very edge as in the engravings, or farther out or back, as the case demanded.

Having described briefly the two forms of apparatus used to obtain traction, I may now speak somewhat in detail regarding their use in practice, and refer to the rules which should govern the employment of the traction.

When we have to deal with the confirmed deformity of especially congenital equino-varus, it must be apparent that, in order to remove the malposition, the force applied should exceed the resistance. No one, I think, would maintain that the skin and subcutaneous tissues could tolerate a force of this nature for any considerable length of time. And experience proves that this is not necessary.

In a lecture on "Knock-knee and Bow-legs," delivered by me in 1879 and published in the *American Journal of Obstetrics and Diseases of Women and Children* (July, 1881), I called attention to the fact that the superficial tissues would tolerate a greatly exaggerated pressure for a few seconds or even moments without any unpleasant consequences, and I made this fact the basis for the mechanical treatment of the conditions there described. And this is the basis of the "Treatment of Club-foot by Traction," whether tenotomy is used or not.

The manner in which the traction apparatus should be used may be briefly described.

The first step is to adjust the apparatus, whether the antero-posterior or lateral, so that *it will exactly fit the deformity*. Then apply the heel traction and astragalus retention straps as described in Fig. 14 and secure the foot firmly in the heel-cup of the apparatus by buckling the astragalus and heel-traction straps. Apply a moderate amount of traction at first, always being sure that the astragalus retention strap is loosened (after the heel-traction strap has been made tense) to permit the upward movement of the astragalus when the ankle is drawn into flexion. Determine at the first application how much traction can be tolerated by the patient without causing pain. Proceed easily and gently at first, and after a few interviews the pressure may be increased to the desired extent without any difficulty or trouble.

After the patient is accustomed to the apparatus, keep the traction-shoe applied most of the time, using gentle traction at first, and removing the apparatus only when the patient goes out of doors for necessary exercise, or at night, when in both instances the proper passive, or retention, support is worn. When this point is gained, commence a systematic application of the momentarily exaggerated traction.

My method is to apply the exaggerated traction at intervals of one half hour or hour for the period of toleration, be it five seconds or five minutes. During the intermission little or no pressure is used, and the patient walks about with the traction shoe applied. Experience proves that what is gained during these seconds or minutes of exaggerated traction is not lost during the intermission, and a frequent repetition of "intermittent treatment" will be followed, as a rule, by a lengthening of the foot, showing that the plantar tissues are yielding; a visible lengthening of the gastrocnemius muscle soon becomes apparent, and the deformity yields to the traction.

Always insist upon careful attention and intelligent home care, and always see the patient once a day for the first few days, after which, in many cases, the mother or nurse can carry out the treatment at home, with occasional visits from the surgeon.

Under these circumstances, and with the use of proper prophylactic measures, as applied to the skin and subcutaneous tissues, excoriations will not occur. In private practice I have never seen any excoriations that interfered with progressive treatment, and in no case have I seen even slight excoriations, except as the result of neglect on the part of the attendant. The traction is tolerated well, the tissues yield, the deformity grows less marked, the muscles increase in circumference, as well as in length, under the influence of the traction, and in very many cases the knife will be unnecessary. We can, in short, restore the muscles and fasciæ, as well as the ligaments, to their normal length, and the ultimate result is a foot more nearly approximating the normal than any which I have ever seen after the usual procedure of tenotomy.

But there are some cases which do not readily yield to traction alone. They are not common, but when they occur it is an easy matter to perform tenotomy and remove the element of muscular resistance. But the patients upon whom tenotomy has been performed complain of pain much more than do those who have been treated by simple traction. In by far the greater majority of cases the pain inflicted by properly applied traction is so slight that neither the patients nor their parents complain. Adolescents and adults bear it without any complaint whatever.

I know of no rule by which any one can determine whether a given case will yield to traction, or whether it will require both tenotomy and traction. It will be a very exceptional case that does not yield to the two combined. My rule is to apply traction first, and, if it fails after a fair trial, to perform tenotomy also. And I know of no apparatus that answers so good a purpose after tenotomy as these traction-shoes. They are readily adjusted, easily applied, and the force can be regulated to any degree of indicated pressure. If tenotomy has been performed, I make a vigorous traction while the patient is still under ether, and gain all I can while the patient is still unconscious. I have even proposed to place a patient under ether and make vigorous traction without tenotomy as a substitute for this operation. But I have not done this yet. The apparatus can be made to exert a great degree of force, and I think I can say that both the antero-posterior and the lateral apparatus are constructed upon correct mechanical principles. Certainly the results obtained by their use warrant me in saying that the prediction I made nine years ago, that the use of traction would "make the necessity for tenotomy much less frequent," has been fulfilled.

I do not oppose tenotomy when necessary. I prefer, as the result is so much better, to obtain the result with traction alone if possible, even if a longer time is taken to produce it. The elongated, freely moving, and symmetrical foot obtained by traction more than compensates for the apparent loss of time. And I have seen no relapse after the tissues are once elongated by traction, as there frequently is after tenotomy.

I have had some failures with this apparatus, without tenotomy, just as I have had many successes. But I am very certain that most, if not all, of the former were due to causes beyond the control of the surgeon, just as I am equally certain that many cases, both among adolescents and children, which have been condemned to tenotomy or even osteotomy by prominent surgeons, have been absolutely restored without operative measures by simple traction, intelligently and perseveringly applied.

FORCIBLE INTERMITTENT TRACTION IN THE TREATMENT OF CLUB-FOOT[1]

Read before the Orthopædic Section of the Tenth International Medical Congress, held in Berlin, August, 1890.

THE phrase "forcible intermittent traction" is intended to describe a simple but effective operation for reducing certain non-inflammatory chronic deformities which the author of this paper introduced to the medical profession in the New York *Medical Record* for November 23, 1878, in a paper entitled "Traction in the Treatment of Club-foot." The subject was further amplified in a paper on "Knock-knee and Bow-legs," published in *The American Journal of Obstetrics and Diseases of Women and Children*, in July, 1881.

The operation, which was referred to in the papers mentioned as "an exaggerated momentary pressure," is based upon the tolerance of the skin and subcutaneous tissues to a very great degree of pressure for a brief period of time, and it is further based upon the well-known law that in order to remove a given obstacle the "force must exceed the resistance." The chief causes which led to the application of these two principles to the reduction of chronic deformity were especially the imperfections in the conventional apparatus for the treatment of club-foot, and the unsatisfactory results that followed their use.

The first thing that I strove to accomplish was to devise an apparatus which could be made to fit any ordinary club-foot at will, with the mechanism so arranged that when the initial force was used it could be directly applied to the resisting tissues. In short, I endeavored to imitate the action of the trained human hand when applied to the reduction of the deformity, and in all of the instruments here described it would seem that not only this object, but even more, has been accomplished, when one estimates the degree of force which the human hand is capable of applying and compares it with that exerted by the apparatus described.

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[1] *The Medical News*, March 28, 1891.

In using forcible, intermittent traction for the reduction of the deformities of talipes, either with or without tenotomy, myotomy, or syndesmotomy, the apparatus is so arranged that the pressure may be carried up to a point of educated tolerance by a gradually increasing pressure, and as soon as the patient indicates that a maximum pressure is reached all force is to be *instantly removed.* If the apparatus is properly adjusted, each forcible traction will be followed by a yielding of the deformity, and if it is applied frequently and systematically, the improvement will soon be very noticeable. If the deformity fails to yield after a sufficient trial, a free subcutaneous section of all the easily reached muscular and fibrous tissues will so modify the resistance that the condition will be a formidable one which does not then yield to this force.

In an article published in the New York *Medical Journal,*[1] March 5 and 12, 1887, *Vide* pp. 375 and 424, I gave some directions for the use of forcible intermittent traction in the treatment of club-foot. Time has demonstrated their general correctness, and I reproduce them here with such modifications as experience suggests.

The first step is to adjust the apparatus *so that it will*

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[1] "The Use of Traction in the Treatment of Club-foot."

exactly fit the deformity. Determine at the first application how much traction can be tolerated by the patient without causing pain. Proceed easily and gently at first, and after a few applications the traction may be increased to any desired extent without difficulty. After the patient is accustomed to the apparatus, keep the traction-shoe applied the greater part of the day, using a simple retention, walking, or a night, apparatus at all other times. By this means the foot will not be permitted to relapse into its former position of extreme deformity.

When the patient learns that he can control the amount of traction, commence a systematic application of the forcible intermittent traction.

The traction should be applied every fifteen or thirty minutes for the period of educated tolerance, be it for one second or five minutes. Patients vary greatly in their ability to bear the pressure, but all may be educated very soon to endure a sufficient degree of pressure to make "the force exceed the resistance" to an extent which shows an appreciable gain in a short time. Experience proves that what is gained during the seconds or minutes of forcible traction is not wholly lost during the period of intermission, and during the latter time the patient may walk about in the traction apparatus, which is adjusted to an easy position with a minimum pressure.

If these rules are followed there will soon be found a lengthening of the plantar tissues, and the gastrocnemius will become visibly longer. In the average case the shortened tissues will yield without the use of the knife; but in some cases, after a few weeks of forcible traction, the resisting tissues seem to yield no further—or, yielding, will, like an elastic cord, gradually shorten soon after the traction is removed. As soon as it is demonstrated that this is the case, all the resisting muscular and fibrous tissues should be divided subcutaneously, when it is probable that one prolonged traction under anæsthesia will remove the deformity.

Excoriations are not likely to occur, and if suitable precautions are taken they do not occur at all. The only case of troublesome excoriations that has happened in my own experience resulted from a too prolonged traction after operation and under anæsthesia.

In using forcible traction without tenotomy, etc., I instruct someone interested in the patient in the use of the apparatus, so that treatment may be carried out at home. This is always very easily accomplished in private practice. The pain inflicted by properly applied forcible traction is so slight that neither the patients, if they are children, nor their parents complain. Adolescents and adults bear it without any complaint whatever.

The question will at once be asked, "What is the length of time required to secure a good result by the use of this method?" My answer is, that if subcutaneous section and forcible traction are used as the initial procedures, the reduction of the deformity can be ordinarily accomplished at one sitting during the anæsthetic stage of the operation. But if a preliminary trial of traction is made, and if the conditions favor its use to the exclusion of tenotomy, etc., the treatment will probably require six months or a year. As is well known, it does not do to dispense entirely with treatment after the simple reduction of the deformity by operation. An apparatus must still be worn, and all that is ordinarily gained by the cutting operation is the immediate reduction of the deformity. At the end of a year the patient who persists in the traction treatment alone will have a much better foot than the one which is subjected to the cutting process. When time is of importance, as it is among some dispensary patients, the rapid process should be employed. When, on the contrary, time is not so important an element, and when the best attainable result is desired by the patient or his parents, it is well to use the forcible traction alone until it is demonstrated that the result cannot be obtained by this means. And when it is stated that several obstinate cases, which were condemned to osteotomy—or amputation—have been cured by forcible traction alone, and without the aid of the knife, it will be seen that forcible traction promises a great deal.

Three forms of apparatus are used: one for the reduction of simple equinus, known as the "antero-posterior traction-shoe"; and two for the reduction of equino-varus: one known as the "internal lateral traction-shoe," and the other as the "external lateral traction-shoe."

The apparatus used is fully described in the article which precedes this—"The Use of Traction in the Treatment of Club-Foot"—to which the reader is referred.

COMMENT BY JOHN JOSEPH NUTT, B.L., M.D., and F.A.C.S., SURGEON-IN-CHIEF TO THE N. Y. STATE ORTHOPÆDIC HOSPITAL FOR CHILDREN, WEST HAVERSTRAW, N. Y.

Having discovered and described the condition which he called "non-deforming club-foot" Dr. Shaffer gave his attention to its treatment by his traction-shoe. I know of no clearer demonstration of the mechanics of the foot and ankle than that made by Dr. Shaffer in showing the effects of a shortened heel cord and the requirements for its correction, which are met in this ingenious apparatus. The direction and range of movement of the various parts of the foot and ankle have never to my knowledge been so scientifically demonstrated and the traction-shoe meets these requirements most excellently.

The mechanical side of orthopædic surgery has undoubtedly been neglected to some extent during the last few years and the operative side has been brought to the foreground. This partially accounts for the lack of a more general employment of the traction-shoe in treating these cases and the resorting to tendon lengthening by the open method.

Dr. Shaffer has pointed out the signs and symptoms arising from the disturbed mechanics of the ankle due to the shortened gastrocnemius and differentiated between these symptoms when arising from slight shortening and when arising from greater limitation of dorsal flexion. I believe that observations which I have since made, but have not yet published, regarding disturbances of the knee, were not studied by him. In many cases of loss of free and painless movements and even in cases of internal derangement of the knee-joint, I have found the gastrocnemius shortened but no important signs or symptoms pointing to foot trouble, the knee seemingly having suffered the brunt of the faulty mechanics. It does this by retaining the knee in a slightly flexed position to make up for the shortening of the muscle and the knee is thereby placed in a position vulnerable to strain especially by lateral movement and rotation. Treatment by the traction-shoe in these cases has proved the correct diagnosis of the basic disturbing factor.

Note by the Author

(September, 1923)

The Antero-Posterior Traction-Shoe has a much wider range of usefulness than appears from its simple construction. Not only is it applicable to chronic sprain and in all cases requiring an elongation of the post-tibial and tarsal tissues, but its usefulness in the toe drop of paraplegia is very noticeable. The immediate result of a few applications is remarkable. In one case it seemed to re-vitalize the muscles of the entire limb, the circulation being notably improved. In another case the patient said that he believed himself to be on the road to recovery, so marked was the improvement. He died of an intercurrent acute disease when he was at his best. It also has been of great service in the treatment of metatarsalgia, the lengthening of the gastrocnemius and plantar fascia gives freer movement to the tarsus and thus modifies the pressure incidental to this condition: indeed a slight lengthening of the post-tibial muscles relieves many of the annoying and painful foot troubles.

The improvement of the circulation of the foot and leg, after a short application of the traction-shoe, is marked. Frequently adult patients with the toe drop of infantile paralysis have visited me in winter saying that after a single application of the traction the foot would keep warm all day.

ON SOME OF THE DEFORMITIES OF THE TARSUS IN CONGENITAL EQUINO-VARUS[1]

WE are all acquainted with the difficulties that sometimes attend the treatment of many of the apparently simple cases of congenital equino-varus. If we regard these cases from a purely objective standpoint, it seems a not very difficult matter to relieve the deformity. The resisting gastrocnemius muscle and the tense plantar fascia offer a well-defined and definitely located resistance to the reduction of the deformity; and our prognosis is too frequently based upon these easily determined elements of resistance, coupled with the degree of mobility that may exist at the ankle and tarsus. But a long record of failures point to the fact that there are some elements of resistance in these cases which cannot always be easily determined,—elements which are beyond the reach of ordinary diagnostic measures, and which have not always been removed by even the most radical operative procedures.

My studies in this interesting field have led me to seek a remedy for some of the difficulties that present themselves in congenital equino-varus, which will insure more uniform and better results; for I feel that all of the cases of this class, if treated before the age of puberty, should recover with straight feet. And I have felt for the past fifteen years that if a properly applied force could be utilized in these cases, very few, if any, would require operative treatment. In brief, I feel that while much ingenuity has been expended in devising and perfecting the various cutting methods of treatment, the apparently simple but really more difficult mechanical methods which promise so much better results have been in many cases neglected.

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[1] *Transactions of the American Orthopædic Association*, Vol. i.

It also strikes me that, as orthopædic surgeons, we take too much for granted,—that there is not enough original research and study among those who practise orthopædy. The anatomy, both normal and pathological, as well as the mechanics of the articulations, claims more of our attention than has been devoted to it. The surgical aspects of chronic deformities are, as we all know, so well taught that it is easy enough to apply the knife, the saw, the chisel, or the osteoclast, if we have occasion to use them. But who is there among us who has completely mastered the subject of the mechanics of the articulations? I cannot help thinking that the next generation of orthopædic surgeons will do less operating than is now practised, and that the much neglected subject of applied mechanics will receive the attention it demands.

There is one popular fallacy in the pathological anatomy of congenital equino-varus that has not received the attention it deserves.

We are taught that there is no lateral motion at the ankle-joint; that the articulation between the inferior articulating surfaces of the tibia and fibula and the superior and lateral articulatory surfaces of the astragalus permits only antero-posterior motion, except in extreme extension. As a consequence we have been taught by many eminent teachers that in congenital equino-varus the principal deformity occurs at the articulation of the calcaneus with the cuboid on the outside of the tarsus and at the articulation of the astragalus with the scaphoid on the inside of the tarsus,—these two transverse joints forming the medio-tarsal articulation.

No one, I think, will deny that some of the major deformities that occur in congenital equino-varus depend upon the changed relations of the bones of the tarsus at or below the calcaneo-astragalar articulation; and no one, I think, will doubt that the medio-tarsal joint plays an important part in the deformities of club-foot. But I wish to assert that the deformity or deformities which occur at the medio-tarsal joint are neither the most important nor the most difficult to remove. If the medio-tarsal deformities were all, or the most important ones, found in equino-varus, my experience leads me to say that our results would be uniformly better.

I do not doubt that anatomists are correct in saying that there is no perceptible lateral mobility at the normal ankle-joint. But it is, I think, absolutely certain, clinically and pathologically speaking, that a very considerable and a very important displacement of the astragalus occurs in congenital equino-varus, accompanied, in some cases, by an intrinsic deformity of the neck of this bone; and I believe that the most important deformity of congenital equino-varus, as well as the most difficult one to remove, occurs at the very point at which many anatomists tell us it cannot occur.

The astragalus is closely held between the two malleoli, it articulates with the calcaneus and scaphoid bones only, and it is the only important bone of the tarsus which is not directly controlled by muscular action. Lying in its doubly protected position, and held in place by its ligamentous attachments, it can only be reached when we desire to correct the position it assumes in the condition under consideration, by attacking the bones with which it articulates. All the other bones of the tarsus are reached with comparative ease, but the astragalus can only be reached by an indirect force.

If under these circumstances we divide the plantar fascia and the gastrocnemius and all the other tissues ordinarily divided in the operative treatment of congenital equino-varus, we cannot, as we all know, insure a reduction of the deformity. We may even make a deep, open incision into the plantar tissues, and cut the anterior portion of the internal lateral ligament of the ankle-joint and divide the tendo achillis without successfully reaching the displaced and rotated astragalus. Still further, osteotomy may be performed and a large wedge removed from the outer side of the tarsus, and still leave the rotated astragalus unmoved in its deformed position. In brief, I believe that the keystone of the deformity is at the misplaced astragalus, and that if we succeed in replacing it in its normal position, the other deformities become of minor importance and are comparatively easily removed.

What is the position of the astragalus in congenital equino-varus? Sometimes, though we cannot always discover it, especially in young subjects, there is an intrinsic deformity of the head and neck of this bone, due, I am quite sure, in the majority of cases to rapid growth during the latter months of intra-uterine life, after the gastrocnemius has become shortened. But whether this intrinsic deformity exists or not, it is apparent that the acquired position of the astragalus in the condition named is not a simple one.

Rather than attempt to describe the malposition of the astragalus relatively in the condition under consideration, I think it better to study its position from a mechanical standpoint. We can then better understand the indications for treatment.

In response to the irresistible force which is acting upon all the misplaced tarsal bones through the medium of the shortened muscular and fibrous tissues, the astragalus (1) becomes rotated upon a *transverse axis;* that is, the head and neck are tilted downwards as it is in the position of extension of the ankle-joint, or as it is in simple equinus. (2) It also becomes rotated upon an *antero-posterior axis*, or its under surface is turned toward the mesial plane of the body upon an axis which corresponds, in general terms, with the long diameter of the foot; and (3) it is rotated upon a *vertical axis*, or the head and neck are twisted toward the mesial plane of the body upon an axis which corresponds with the long diameter of the tibia, aiding in the production of the so-called inversion, or adduction of the tarsus. In a typical case we find all these rotatory elements of deformity present, not only at the astragalus, but also at all the other tarsal bones, producing a compound deformity that so disturbs the normal line of gravity that the patient in walking presents the cuboid bone to the floor.

A brief study of the anatomical construction of the tarsus, bound together as its component parts are by strong ligaments, will, I think, convince any one that the extreme malposition of the cuboid bone (as a matter of illustration), and which is seen in all extreme cases of equino-varus, could not occur except as a result (1) of a dislocation at the calcaneo-cuboid articulation, or (2) a dislocation at the calcaneo-astragalar articulation, or (3) a malposition of the entire tarsus, accompanied by a greater or less elongation of all the ligaments on the concave side of the deformity, and a changed relation of all the articular tarsal surfaces. It is known that there is no true dislocation of any of the bones of the tarsus under the conditions named. The articular surfaces are disturbed, subluxation to a minor degree is present, and interarticular pressure is concentrated at certain exposed points. It seems evident in these cases that there is, among other things, a loss of relative growth between the muscles and the bones, especially the long bones of the leg, and that the arrested or impaired growth of the gastrocnemius produces a malposition that prevents the foot from assuming a normal position. Growth of the bones, however, goes on; and the result is that the tarsus, like every other complex body, yields in the direction of the least resistance. As the tarsus yields in response to the irresistible force of opposed bony growth, the os calcis, the cuboid, and the scaphoid are pulled, so to speak, from their normal position, dragging the astragalus with them, after their ligaments have been stretched to their utmost capacity. In short, a certain degree of displacement of the astragalus takes place; and this is largely due to the fact that the superior articular surface of the astragalus as well as the lateral facets are free from ligamentous or muscular attachments, and also because the ligamentous attachments of the lateral as well as the anterior and posterior surfaces of this bone are low down, or *below* the centers of rotation to which attention has been called. There is, therefore, no ligamentous or direct muscular resistance to the rotation of the astragalus when the os calcis, the cuboid, and the scaphoid are drawn from their positions. The ultimate point of resistance lies, therefore, at the inferior articular surfaces of the tibia and fibula; and as a result of the great and prolonged pressure which exists, it may be presumed that absorption of the articular cartilage ensues; and this, with the slight rotation of the fibula upon the tibia upon their long axes, accounts for some of the lateral or rotatory movements which the astragalus undergoes in the deformity under consideration. Even absorption of bone may follow; and it is not impossible that the ligamentous structures which bind the fibula and tibia together may yield as the result of the long-continued pressure, or that, especially in fœtal life, the astragalar neck may become twisted.

Associated with the shortened gastrocnemius in congenital equino-varus, we always have a shortening of the plantar tissues. This plantar shortening fixes the calcaneus in a position where it cannot rotate outward, this latter condition occurring only, as a rule, in flat-foot with elongated plantar tissues, or in valgus with a paralysis or weakness of the gastrocnemius. An important feature of congenital equino-varus is therefore an inward rotation of the calcaneus upon an antero-posterior axis, associated with a rotation upon a transverse axis.

After the remarks made upon the nature of the muscular shortening we will assume that the normal longitudinal growth of the gastrocnemius is either arrested or impaired, and that the tibia is not affected to the same extent by the lesion or change which produces the so-called contracture of the muscles. If the tibia outgrows the muscle and as a result of the irresistible force produced by longitudinal osseous growth in the long bones of the leg, a deformity must occur somewhere. In congenital equino-varus it is first seen in the rotation upon a transverse axis and in the complex rotation of the os calcis already referred to. Perhaps the tibialis posticus is also affected, as I think it is in many instances. The plantar tissues certainly are shortened. With these elements at work, the deformity of equino-varus is not difficult to explain from a mechanical standpoint. The whole tarsus is rotated like the keystone (the astragalus) upon the three axes already mentioned. The hinge of this rotation, so to speak, is at the point of the least ligamentous and muscular resistance, the tibioastragalar articulation; and as a result of the prolonged and uninterrupted pressure, the whole tarsus assumes a position of chronic deformity, the various articulations becoming more rigid as time passes.

It is well to stop here for a moment to point out the conditions which the ligaments of the tarsus assume under these conditions. On the outer aspect of the foot they become elongated; on the inner they soon become very materially shortened. This is especially so at the internal lateral ligament of the ankle-joint. All the other ligaments become either lengthened or shortened, depending upon their location and their relation to the acquired position of the bones to which they are attached,—those on the convex side being elongated, those on the concave side being shortened.

After a time,—but I think after a much longer time than is generally supposed,—the articular surfaces of all the bones involved adapt themselves to the deformed position. My experience leads me to think that it is exceptional for the osseous troubles to reach a point of more than troublesome resistance before puberty, unless there is a considerable intrinsic deformity of the neck of the astragalus; while the ligaments on the other hand become very much shortened, even in babyhood.

I feel, therefore, that we may assume the following as a basis to treat the condition under consideration: First, that there is a compound deformity in equino-varus, due to a rotation of all the bones of the tarsus upon three distinct axes,—that is, transverse, antero-posterior, and vertical; secondly, that this compound deformity is due to a loss of relation between muscular and osseous growth, —not to muscular contraction or contracture, as has generally been supposed; thirdly, that the principal obstacles to a reduction of the deformity are (1) the position acquired by the astragalus with or without an intrinsic deformity of the neck and (2) the ligamentous shortening; fourthly, that these difficulties are much increased by the progressive ligamentous and osseous changes that occur as the direct result of prolonged malposition; and fifthly, that the deformities of the medio-tarsal joint are secondary in importance to those which occur at the astragalus.

We will now pass to the question of treatment.

In undertaking any plan of treatment we must bear in mind that the whole tarsus is involved, and that any method which restores the astragalus to its normal position promises to be successful; for if we succeed in restoring the normal relations of the astragalus to the tibia, we carry with it (the astragalus) the other bones of the tarsus. My experience proves that it is not the antero-posterior deformities, where rotation occurs upon a transverse axis only, that present difficulties. The real difficulties occur in the so-called "lateral" deformities, where we see the effects of the compound rotation to which attention has been already directed. In order to make myself better understood, I will again briefly refer to the three varieties of rotation: the first-named, the *transverse*, is shown at the elevated heel; the second, the *antero-posterior*, is evident at the inward rotation of the os calcis; the third the *vertical*, is seen in the inversion of the entire foot.

The result of the first-named condition, as has already been stated, is easily overcome if it be uncomplicated. The second and third conditions are due largely to the causes which produce the first, and may be considered as secondary to it.

Our first effort, therefore, should be to attack the gastrocnemius shortening in such a way as to overcome its secondary effects; and it must be evident that any method which neglects the gastrocnemius shortening must fail. But the entire tarsus is rotated, and we must not make the error of using the cuboid as a point of resistance to overcome the rotation of the tarsus, for the simple reason that if we do so we at once block the mechanism, whatever it may be, designed to overcome the rotation of the tarsus. The cuboid has taken its acquired position on account of its close relation with the os calcis. The tarsus as a whole should be thrown *downward* and *outward* (reversing the curve of the deformity, which is *inward* and *upward*) on an antero-posterior axis; and this downward and outward rotating force should be applied upon a remote axis until the os calcis and the astragalus fall directly under the tibia, the mechanism being so arranged that the rotation upon a transverse axis is also attacked. We must also remember in treating all these cases, even those seen immediately after birth, that we are not dealing with muscular shortening only. Two other potent factors must be considered; and these are, first, the resistance which exists at the shortened ligamentous structures, and, secondly, a possible or even probable intrinsic deformity at the neck of the astragalus. And it must be remembered that these ligamentous structures soon adapt themselves to the acquired position of the bones of the tarsus. It is surprising how soon these ligaments shorten, and this remark is especially true of the internal lateral ligament of the ankle-joint. Any procedure which ignores this condition or the changed articular surfaces is apt to result in failure; and the difficulty of attacking these shortened ligamentous tissues, especially those upon the internal malleolar aspect of the ankle, is apparent. It is very easy to apply traction to the antero-posterior contractions, of whatever nature; but it has not been so easy heretofore to reach the internal lateral ligamentous contractions or the twisted astragalar neck by this same method of traction, though it seems evident that the resistance of a contracted ligament or a deformed bone is just as important as that of a shortened muscle, and much more difficult to reach. And this principle of lateral traction should not be applied without recognizing the fact that the cuboid bone must rotate downward and outward with the other bones of the tarsus upon a remote center of motion in removing the deformity of equino-varus. My experience has proved that if the whole foot is rotated outward upon a remote center by an adequate mechanism, the heel being properly secured, the major deformities become modified very soon.

In my experience the principal obstruction to the reduction of the deformity (excepting, of course, an intrinsic deformity of any of the tarsal bones) exists in the shortened ligamentous tissues which lie between the inner malleolus and the tarsus. I formerly regarded the plantar tissues as being the most obstinate, and they still tease us all. But the internal lateral ligament of the ankle is very strong and dense. It arises from the internal malleolus, and sends its radiating fibers not only to the os calcis and scaphoid, but to the astragalus as well. This ligament is always shortened in congenital equino-varus, and the tarsal bones to which it is attached are imprisoned in their acquired positions until the contracted positions of the ligament are elongated. I regard this ligamentous resistance as being far more important than the gastrocnemius or plantar shortening. If we can find some means to overcome this shortening of the internal lateral ligament, I am sure we will find that the duration of the treatment of congenital equino-varus will be very materially shortened, and much better and far more permanent results will be secured.

The apparatus I will show you on this occasion treats the internal, lateral, and other ligamentous resistance exactly as the gastrocnemius is treated in our efforts to overcome the antero-posterior contractions of equinus with the antero-posterior traction-shoe.[1] The aim of this new traction apparatus is to bring the rotated tarsus, as a whole, and by a very simple movement, directly under the inferior articulating surface of the tibia, attacking not only the post-tibial resistance, but also and especially the stubborn shortening of the internal lateral ligament, as well as all the other contracted tissues upon the inner or concave side of the deformity. If the neck of the astragalus is twisted, a very great force is applied to it in the right direction. I had previously attempted to accomplish these ends by a pushing force acting from the inner side of the foot (the internal lateral traction-shoe). But experience proves, in a very limited number of cases, that it is difficult to apply the rotating force accurately when using this internal lateral stretcher; and it was also found that the rotation of the cuboid was sometimes arrested by the mechanism of the apparatus before it fell into its proper position. And it may be that a sufficient force could not be obtained in cases where there was an undiscovered twist at the neck of the astragalus,—a criticism which cannot be made of the external traction-shoe, which can be made a "crusher" if necessary.

In the apparatus I now show you, while sufficient pressure is exerted upon the cuboid, by a simple retention strap, to make it a simple point of resistance, the apparatus is so arranged that the whole tarsus is rotated *downward* and *outward* to any desired extent; and a direct pressure is exerted in the right direction upon the twisted astragalar neck, if any deformity exists at that point.

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[1] Vide, "The Use of Traction in the Treatment of Club-Foot," *New York Medical Journal*, March 5 and March 12, 1887.

In Fig. 1 there is shown a schematic engraving of the external lateral traction apparatus, illustrating its application to a condition of equino-varus of the left foot, and its movement upon a remote antero-posterior axis. The center of motion is at the point A, remote from the tarsus and outside of, but directly over, the external malleolus. The anterior portion of the foot rests upon the foot-piece B, inside of the curved lateral plate C, which grasps the inner aspect of the foot at the junction of the first metatarsal bone with the first phalanx of the great toe. The heel is retained by the conventional astragalar retention strap, and the heel traction-strap of the antero-posterior traction apparatus. The foot-plate of the apparatus is removed posterior to the point which corresponds with the junction of the tarsus and metatarsus, leaving an open and un-

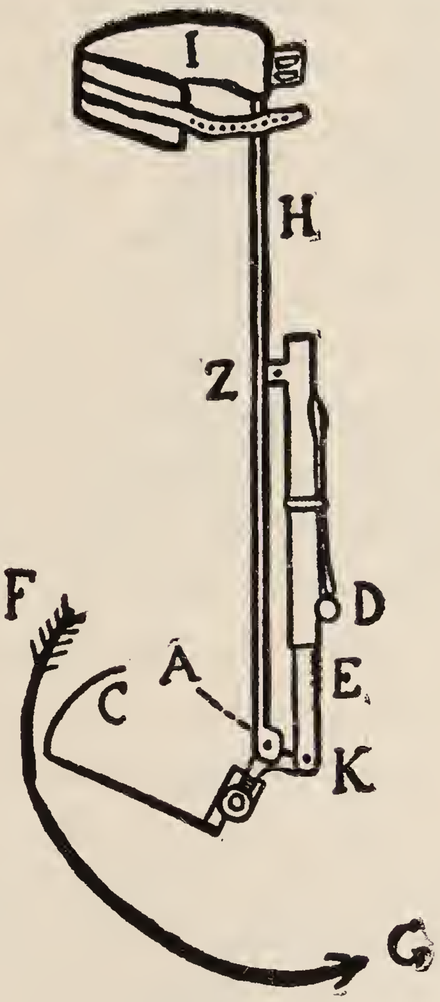

Fig. 1

obstructed space for the descent of the heel where traction is applied. The necessary arrangements to secure the heel, etc. (not shown in this engraving), are the same as those used in the application of the antero-posterior traction-shoe which is described in full on pages 375 and 424.

The foot being secured by the straps referred to, in the apparatus now under consideration, in its deformed position (at the angle, for example, shown in Fig. 1), a key (A, Fig. 2) is introduced at the cylinder D and the extension rod E is *shortened.* The entire foot-piece of the apparatus is thrown downward and outward by this movement, in the direction of the curved arrow F, G, upon the remote center A; and the foot, properly secured in the apparatus, follows the same movement.

The foot mechanism is attached to a straight, strong steel bar, H, and this side bar is surmounted by a conventional calf band I, which is made to closely encircle the calf at a point just below the knee-joint. The *retraction* rod is attached to the side bar at L. In varus this side bar, H, is placed on the outside of the leg; in valgus on the inside. In difficult cases this side bar may be continued upward so as to include both leg and thigh, with a perineal pad to afford a point of counter-resistance to the force applied below. But in any event, and in order to secure a proper adjustment of the apparatus and to exert an adequate force in the right direction, the leg piece of the side bar, H, should lie directly over the fibula, and at least one inch away from it at the external malleolus in the treatment of equino-varus.

Fig. 2 gives a side view of the apparatus, and illustrates the action of the apparatus upon a transverse axis.

The centre of motion on a transverse axis is at the point B; the key C acting on a worm and screw which lowers or elevates the anterior part of the apparatus D. The foot being retained in the apparatus by the astragalar and heel retention straps already referred to, and secured in the exact position of the deformity, the initial force is applied by turning the key C so as to move the anterior part of the apparatus D in the direction indicated by the curved arrow E, F, until a considerable resistance is offered by the shortened tissues of the foot. Then the key A is used, the outside *retraction* bar and rod are forcibly shortened, and the whole foot rotates downward and outward as indicated by the curved arrow F, G, in Fig. 1.

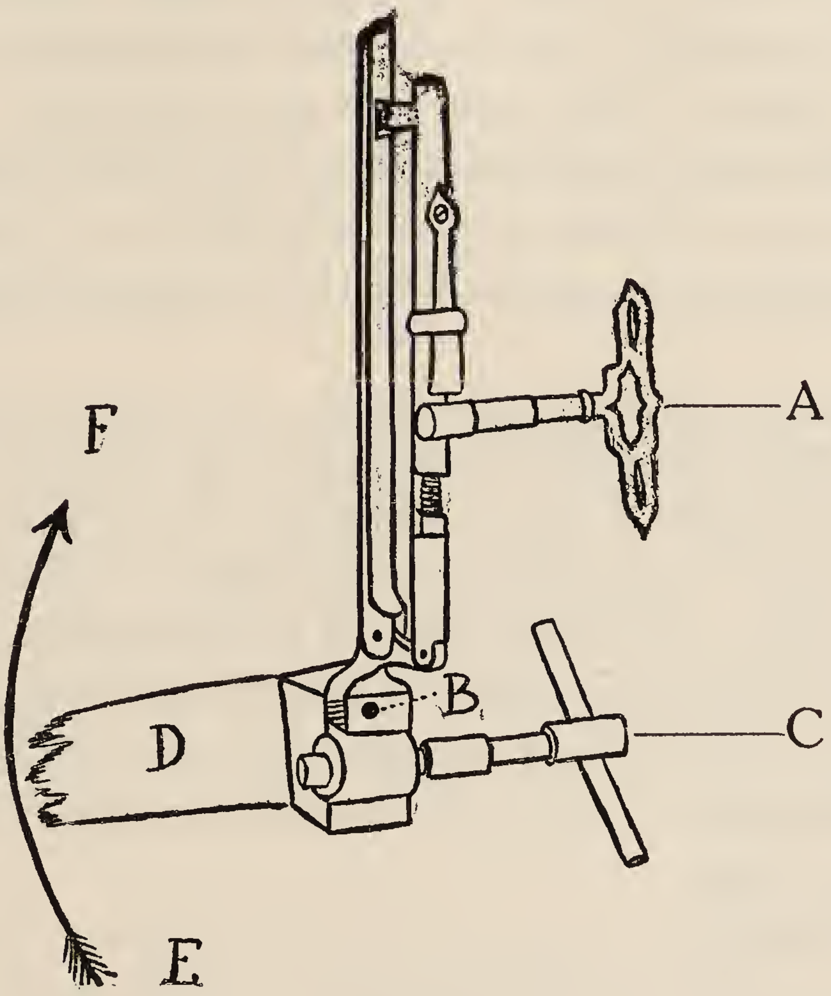

FIG. 2

The curved lateral plate C (Fig. 1) can be moved at will and secured by a key and clamp in any desired position, thus meeting any ordinary amount of inversion of the anterior portion of the tarsus. In short, it is not ordinarily necessary to make any special provision for the rotation of the tarsus upon a vertical axis. When, however, any special mechanism is necessary, that attached to the foot-plate of the "internal lateral traction apparatus" (described in full in the *New York Medical Journal*, March 5 and 12, 1887) meets all the indications. In some cases I have used this attachment with advantage; but in almost every case the vertical rotation has yielded to the combined force represented in the accompanying engravings.

In ordering this apparatus, the center (A, Fig. 1) can be placed at any desired point; and its position will vary with the extent of the deformity, the amount of force required, etc. The same remark applies to the arm (A, K, Fig. 1), which can be lengthened or shortened as occasion demands. It is easy to see that with the combined force exerted by both these powerful levers an osteoclast could easily be constructed, and I am now having one made with this end in view.

I am indebted to my friend Dr. Charles L. Scudder, of Boston, for his assistance in the preparation of the engravings which accompany this paper, as I also am for his recent contribution to the etiology of club-foot.

REMARKS ON THE TREATMENT OF CLUB-FOOT BY OPEN INCISION[1]

Read before the Surgical Section of the Academy of Medicine on the occasion of the discussion of Dr. Phelps's paper, December 12, 1887.

THE fact that the treatment of club-foot by open incision is advocated very strongly by Dr. Phelps, and is practiced almost exclusively by him in a class of cases described as "inveterate," coupled with the fact that there are a considerable number of surgeons who maintain that even tenotomy, in the same class of cases, is frequently unnecessary, leads one to think that the principles underlying the treatment of this deformity are not yet fully understood. And no rule has yet been formulated which affords us the least aid in deciding between these two extremes. It seems as if there ought to be some safe medium course.

It has been my own experience to see many cases which Dr. Phelps would describe as "inveterate" yield completely and even rapidly to simple traction, and I have seen apparently simple cases, which promised to yield to traction, require operative measures. If we could say positively and definitely when a club-foot presents to us for treatment that open incision was necessary, or that osteotomy would be required, or that traction with or without subcutaneous tenotomy or syndesmotomy would answer, we should be much nearer the solution of a difficult problem. But surgeons look at this matter from widely different standpoints, and, depending upon their education and their successes and failures with the conventional methods, they are inclined, I think, to adopt a pessimistic view when they contemplate the indifferent results so frequently obtained by the methods which have been very generally taught since the days of Stromeyer.

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[1] *The New York Medical Journal*, for December 31, 1887.

I think I can very safely say that it is quite exceptional, in both my private and public practice, to find a case of congenital club-foot which has not been operated upon once or oftener before it came under my care. It seems to be safe to say that *failure is the rule after tenotomy and the use of conventional methods;* or, to put the matter in other words, as success is not very often obtained by the methods advised in our standard works, it is not surprising that osteotomy and open incision in the treatment of club-foot should have been among the developments of modern and progressive surgery.

My experience leads me to say that, in the great majority of cases, neither open incision nor osteotomy is required. And I base this opinion *not* upon the failure of *others*, but upon my own failures and successes in my attempts to test and perfect a new method of mechanical treatment based upon a study of the mechanics of the normal foot, and a further study of the changed relations which occur in the conditions under consideration. In making these tests, and in estimating the effects of properly applied traction, I have used my apparatus in some instances where there was more than a legitimate doubt as to its propriety, and have failed on some occasions; but in some cases that had been condemned to osteotomy or open incision, and in one case where amputation was proposed, perfect results have been obtained without even tenotomy by the use of the simple traction apparatus.

These results—failures and successes—were secured while I was experimenting, and during that which might be called provisional work. I feel now that I can crystallize my experience somewhat, and formulate it so that it may, perhaps, be of benefit to others

We can divide congenital club-foot into three distinct varieties, as applied to the question of surgical and mechanical treatment: (1) *Muscular club-foot*, when there is little or no ligamentous shortening, and no considerable change in the bones of the tarsus; (2) *ligamentous club-foot*, when the ligaments are the principal opponents to a reduction of the deformity; and (3) *osseous club-foot*, when the chief obstacle lies in a greater or less deformity of the tarsal bones, but principally in the astragalus.

Can we distinguish clinically between these three conditions? I am sure we can not always do so. Even if we could, I am prepared to say that the extreme measures of osteotomy and open incision would be called for very rarely. We can not determine, even in a case described by Dr. Phelps as "inveterate," that the cause of the deformity can not be removed by simple traction, or by traction and subcutaneous tenotomy, or by traction, subcutaneous tenotomy, and subcutaneous syndesmotomy. We can perhaps gauge the muscular resistance, but we can not duly estimate either the ligamentous or the bony resistance until after we have either elongated the muscular tissue by stretching it, or gained position by lengthening the tendons by cutting them; and if the muscular resistance, and especially if the muscular and ligamentous resistance, has been removed subcutaneously, the condition is a very rare one (under the age of fourteen years) in my experience (the same opportunity being given for observation that exists after open incision has been performed), that does not yield to subsequent traction.

I think it a safe rule to make the diagnosis of muscular ligamentous, or bony club-foot by exclusion. Use an efficient traction apparatus (which is the best one I know of to follow up subcutaneous tenotomy and syndesmotomy) for a sufficient length of time to determine whether the deformity will yield to traction alone. It will do so in many so-called "inveterate" cases. If traction fails divide the tendons and ligaments subcutaneously, and follow up the operation by vigorous traction, applied systematically, as laid down in my paper on "Traction,"[1] already published.

If these rules are consistently followed, I think even the most skeptical will be convinced that "open incision" and "osteotomy" are operations very rarely called for in the treatment of club-foot.

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[1] See *New York Medical Journal*, March 5 and 12, 1887, "The Use of Traction in the Treatment of Club-Foot." Vide pp. 375 and 424.

FLAT-FOOT: ITS CAUSE AND TREATMENT [1]

The substance of the following remarks was made by the author of this paper at the first meeting of the American Orthopædic Association, held in New York in June, 1887. The essay itself was read at a meeting of the Orthopædic Section of the New York Academy of Medicine, held February 17, 1893, and repeated at the meeting of the American Orthopædic Association held in St. Louis the same year.

In order to present the results of my studies in flat-foot in the briefest manner I will call your attention to Fig. 1.

In it I have attempted to convey schematically that which may be easily demonstrated with the recently prepared anatomical subject, or upon the skeleton—namely, the action of the flexors and extensors upon the ankle-joint, the tarsus, and the plantar tissues; the axis of motion at the ankle-joint, with the arcs of motion about this axis of the heel, the superior articulating surface of the astragalus, the medio-tarsal joint,[2] and the distal end of the metatarsal bones.

In this diagram (Fig. 1) A represents the tibia and fibula; B, the knee-joint; C, the origin of the gastrocnemius muscle at the femur; D, the gastrocnemius, soleus, and plantaris; and E, their insertion at the os calcis. In front, B represents the origin of the flexors of the ankle; F, the flexor muscles; and G, their insertion at the tarsus. H represents the plantar tissues.

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[1] *The New York Medical Journal* for May 29, 1897.

[2] In order to avoid confusion and multiplicity of words, the phrase "medio-tarsal joint" in this paper will include all the articulations of the tarsus anterior to the astragalo-scaphoid and os calcis-cuboid articulations.

No attempt is made to show the lateral muscles, for their action upon the ankle-joint center of motion is very limited, and they play no important part in the causation of flat-foot.

Having described the various parts which are schematically apparent in the diagram, I will now call attention to the mechanism.

The point I represents the transverse axis of motion at the ankle-joint. This transverse axis passes through the astragalus at a point just below the external malleolus. The curved line K′ K represents the superior articulating surface of the astragalus, over which the tibia and fibula ride. The center of motion of this curve is at I. The curved line R′ R represents the arc of motion of the distal end of the os calcis, as it moves around the center I. The curved line M′ M shows the arc of the head of the astragalus on the center I; and the curved line T′ T at the distal end of the first metatarsal bone shows the arch of the anterior part of the foot. Its center is traced to same point I, around which all important ankle-joint movements must occur.

The sole of the foot—*i.e.*, its outer border—is represented in all these diagrams at ninety degrees with the long axis of the tibia. If the post-tibial extensors (D) are shortened, the heel (E) is drawn up, as shown by the curved line E R, the superior articulating surface of the astragalus follows the curved line K, and the medio-tarsal joint—at least the astragalo-scaphoid part—follows the curved line M M.[1] Following the diagram forward to the point S (the metatarso-phalangeal articulation), this articulation would follow the curved line T. If there were no medio-tarsal joint at L, S would move on the same center, I, only movement here is modified by this joint L, as will be shown in another diagram.

The one thing which is the principal stay of the arch formed in the foot, and which controls the relation of G to E (the heel and the metatarsus), is the plantar fascia—the bowstring H. This bowstring is tightened when the flexors are shortened, or when the weight of the body

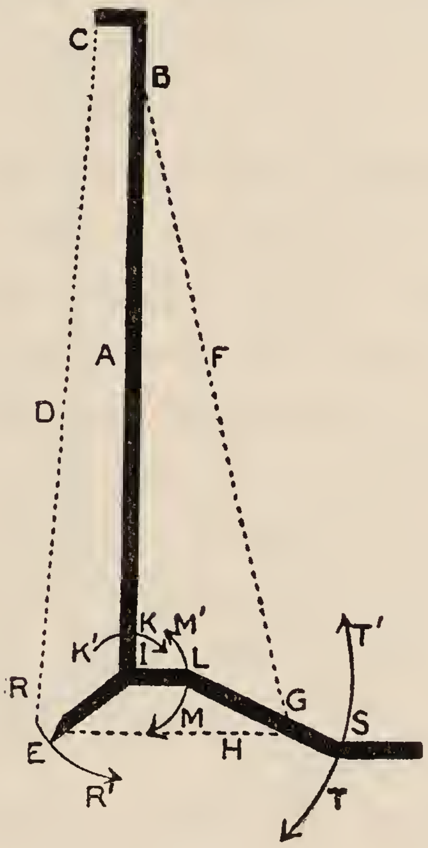

Fig. 1

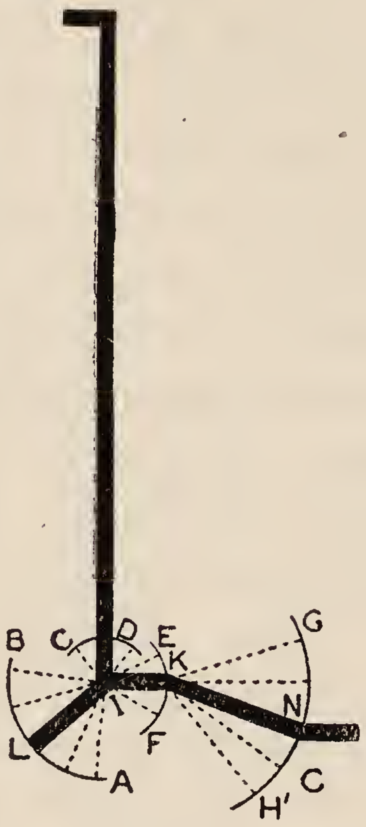

Fig. 2

is thrown on the foot anterior to the medio-tarsal joint. It is simply made taut by the action of the flexors. The strain upon it is more severe when it is called upon to bear the weight of the body with shortened extensors.

In Fig. 2 the same principles are more concisely shown. I again represents the transverse axis of motion at the ankle-joints. The curved line B A is the arc of motion of the os calcis; C D the arc of motion of the superior articulating surface of the astragalus, and E F the arc of motion of the head of the astragalus. These curves all have one common center, I, but each has a radius of different length. In this diagram the movement of the foot anterior to the medio-tarsal joint is shown. The dotted lines K G and K H show the radii of this arc, motion occurring at the medio-tarsal joint K upon the curved line H G until motion is blocked by the plantar tissues, when the center of motion is transferred to the center I, as is shown in diagram No. 1.

There are only two important centers of antero-posterior motion in the human foot—viz., I at the ankle and K at the medio-tarsal joint. The center K is situated between the center I and the distal part of the foot. The weak point of the structure is not at I, for it lies strongly protected in the dense structure of the astragalus, and it can only be disturbed by a force that would rupture or stretch the ligaments binding the os calcis and astragalus together, whereas K is protected only by the plantar bowstring H (Fig. 1). The os calcis and astragalus form, in effect, one bone, very slight motion only occurring between them. There is no weak point in its structure. Motion occurs upon the three remote arcs mentioned, and there is no joint at the axis of motion.

If force is applied at L (Fig. 2), either in flexion or extension, it passes around the remote arc, H G, and is lost first at E K F, and when motion is checked at K it is then found at G N H. Extension of the ankle at L B would produce K F, and if there were no plantar check, K F would form a very considerable arc of motion. If H (Fig. 1) be for any cause weak or imperfect in its function of protecting the medio-tarsal joint K, motion at G H is very materially increased, especially in the direction N G.

In Fig. 3 the principle is shown more fully, and is applied more exactly to show the effect of the weight of the body upon the plantar tissues in locomotion.

If A, the distal part of the first metatarsal bone, is placed upon the fixed point B, and the gastrocnemius D is shortened in the direction of the arrow, the heel moves toward R, the superior articulating surface of the astragalus moves toward K, and the head of the astragalus moves toward M. After the movement at L is checked by the plantar tissues H, this bowstring is stretched, as shown by the arrows in its course. If H is strong enough to resist the force applied, the inner border of the foot does not reach the floor.

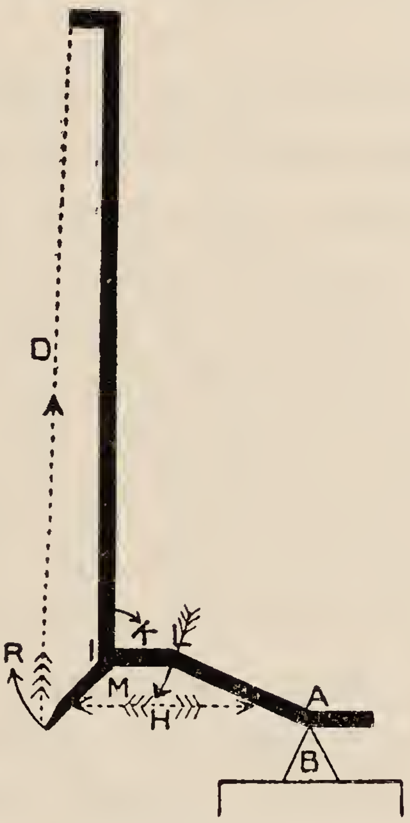

FIG. 3

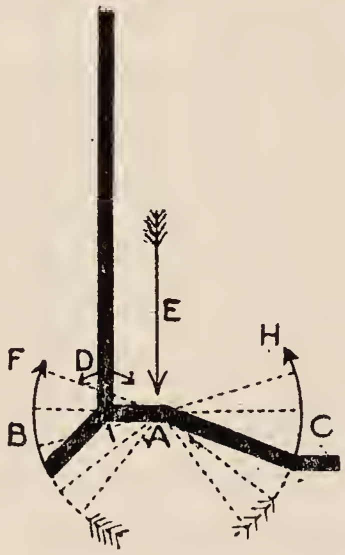

FIG. 4

In Fig. 4 this proposition is still more simplified. We will suppose motion to have occurred at the ankle center, I, until the plantar bowstring has reached its limit, and that the anterior portion of the foot is resting upon a fixed point, as in Fig. 3. Motion then occurs around the center A (the medio-tarsal joint), and the weight of the body falls upon this joint in the direction of the arrow E.

I do not think that any one who has studied the mechanism of the human foot closely will dispute the correctness of these demonstrations, and without them a study of flat-foot would be incomplete.

There is no lateral movement at the normal ankle-joint, unless, perhaps, there may be some slight movement in this direction in extreme extension of the ankle-joint. Whatever purely lateral movement occurs is found anterior to the distal articulating surfaces of the astragalus and os calcis.

The lateral movement of the anterior part of the tarsus is controlled largely by the peroneus longus and peroneus brevis, and by the tibialis posticus. We should not need to consider these muscles in a study of flat-foot were it not that they are also feeble extensors of the ankle-joint; for it can only be the extensors that can raise the heel and produce the effect shown in the diagrams. The tibialis posticus may be dismissed from our minds, as its action is to adduct the anterior part of the tarsus, and it is the legitimate antagonist of the two peronei whose function is to abduct the same part. I do not think it is possible for the unaided action of the two peronei to produce flat-foot any more than the flexors could produce this effect. Under the influence of a tarsal (osseous) inflammation the peronei may assume a certain re-enforced function, as represented by a condition of reflex muscular spasm, and thus abduct the anterior part of the tarsus. But the cause of the increased action of the abductors would be the inflammation. And I might as well say here that the so-called inflammatory flat-foot, with reflex spasm of the peroneal abductors, always represents an advanced stage of flat-foot after a long-continued use of the overstretched plantar tissues has produced a traumatic osteitis in the tarsus. We must look farther and deeper for the cause of flat-foot than the conditions found in advanced cases.

We must try not to mistake effect for cause in our investigations.

If the ankle-joint were like the knee, hip, shoulder, or elbow, we should not have such a condition as flat-foot. In these major articulations all the movements of the joint are represented by a simple lever, with the fulcrum at the joint. The force of the constantly applied muscular antagonism in walking, as well as the weight of the body at the hip and knee, is directly expended on the articulation used. But between the insertion of the flexors and extensors of the ankle there is a series of joints inserted (represented in the paper by the phrase medio-tarsal joint) with a protecting band (the plantar tissues), and it is at these points—the medio-tarsal joint and the plantar bowstring—that we must look not so much for the cause of flat-foot, as for the effects of the peculiar and complicated mechanism which makes flat-foot possible.

The plantar bowstring is at once the weakest and strongest part of the foot. With all the force that has been applied to it in the many accidents that have befallen the ankle and tarsus, the plantar fascia is not likely to rupture like the tendo Achillis. On the other hand, if exposed to a long-continued strain, under certain favoring conditions, it is very apt to elongate. Still, again, if the normal use of the plantar tissues is neglected for any cause, they will shorten and become very dense and hard. It is important to remember that this plantar bowstring may be lengthened if necessary, just as it may be made shorter if given prolonged rest under favorable conditions.

The normal balance between the flexors and extensors of the ankle-joint and the abductors and adductors of the anterior part of the tarsus in the normal foot is usually well preserved. The balance between the post-tibial muscles and the plantar tissues is also generally not disturbed by ordinary circumstances. The balance between the flexors and the plantar tissues is never disturbed by the unaided flexors themselves—any more than it could be by the unaided action of the lateral muscles. The weight of the body in standing or in locomotion must be added to the flexor action, or the peronei must be aided by the instinctive muscular spasm before any pronounced effect can be looked for at the plantar bowstring; and first, and before all, there must be some block to the antero-posterior ankle-joint motion in the direction of flexion before any decided effect can be found at the plantar bands.

If, however, the normal antero-posterior motion at the ankle-joint is disturbed, certain results are very sure to follow. If the flexors are paralyzed, as in poliomyelitis anterior, the post-tibial muscles shorten, and they may shorten so much that the normal use of the plantar fascia is modified or abrogated. The plantar fascia then becomes in effect a continuation of the gastrocnemius, and being unopposed by the normal antagonism of the flexors, and being deprived of its normal use in locomotion, it shortens. If the peronei are paralyzed also, we have a marked shortening of the plantar bowstring, and equino-varus results. If the tibialis posticus is paralyzed, we have ultimately an equino-valgus (an exaggerated flat-foot), with lengthened plantar tissues and an abduction of the foot, which in the end becomes a flat-foot. If the post-tibial muscles are paralyzed, the heel is drawn forward into the calcaneus position. The unimpaired plantar muscles and tissues now become a prolongation, so to speak, of the contracting flexors. The os calcis is drawn forward beyond its normal position, the plantar tissues shorten very much, and calcaneo-cavus results. A condition which is exactly the reverse of flat-foot now occurs.

When the heel is raised to any considerable extent, as in equinus, the weight of the body is transmitted, in walking or standing, through the ankle, tarsus, and metatarsus, in a line which, in extreme cases, approximates the long diameter of the tibia, as is shown in Fig. 5. A A represents the foot at ninety degrees with the tibia; B B, the foot in extension. The dotted line H represents the plantar tissues in the ninety-degree position, while the line N shows the plantar tissues in the extended position.

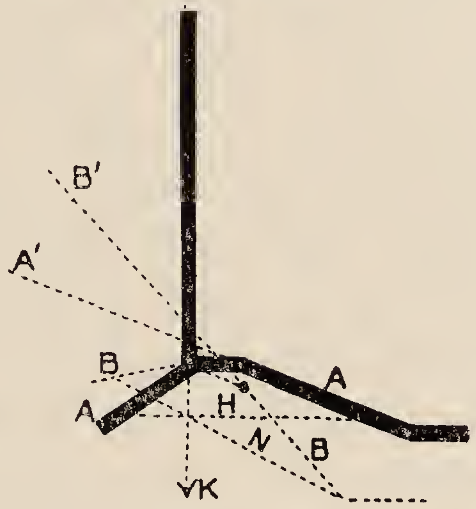

Fig. 5

K shows the force transmitted through H and N, the former forming a right angle with H, the latter an obtuse angle, anterior to the medio-tarsal joint. The strain is much greater in the position A than B, and the extended lines A′ B′ show the direction of the metatarsal bones and their relation to the long diameter of the tibia. The strain upon the plantar tissues is very much modified when extension occurs beyond a certain point. Hence, when the heel is raised to a considerable extent, flat-foot is not likely to occur; on the contrary, the plantar tissues are likely to become shorter, as the normal strain of locomotion upon them is modified. When the post-tibial tissues are shortened only slightly, as in non-deforming club-foot, and the plantar tissues are normally strong, flat-foot is not likely to occur. In non-deforming club-foot, the tarsus is, as a rule, protected by the tibialis posticus, and the weight falls on the outer border of the foot. Non-deforming club-foot generally presents a dense and shortened plantar fascia, and its cause is probably some remote trophic central lesion accompanied by the reflex irritability which Sayre has pointed out. Not a few of these conditions result in flat-foot, however, and they are most frequently seen associated with cases of rotary lateral curvature in young and growing girls. Nature can stretch the tissues that Sayre's dogma says we must divide. On the other hand, if we have a slightly shortened gastrocnemius with weak and flabby fibrous and muscular tissues at the plantar arch, it is only a question of time as to when the bow-string will yield and flat-foot result. If the weight of the body falls squarely on the astragalus, with normal antero-posterior motion around the astragalar curve, there can be no undue strain upon the plantar tissues in locomotion. The weight of the body is diffused around the astragalar curve, and there is no shock or strain upon the plantar bowstring.

Let us suppose that we have a shortened gastrocnemius muscle in a weak or poorly nourished and rapidly growing adolescent. The heel can not quite come down and thus allow antero-posterior motion at the ankle around the astragalar arc. The weight of the body is not sufficient to overcome the post-tibial resistance. The anterior tibial (flexor) muscles are not strong enough to remove it. The lateral muscles are feeble extensors. They would assist rather than retard the stronger extensors. But still the patient walks, and perhaps must walk, or, worse still, stand for a prolonged period to earn his living. The plantar bowstring can not withstand the constant strain. The foot and ankle structures do as other structures do when exposed to a prolonged strain—viz., they yield at the weakest point. In brief, since motion can not occur at the transverse ankle center of motion, the plantar tissues yield, and vicarious motion occurs at the medio-tarsal joint.

As the plantar tissues yield to the shortened extensors and the superincumbent weight of the body, the inner border of the foot becomes longer, the medio-tarsal joint on its scaphoid side falls, and the anterior part of the tarsus passes necessarily into abduction.

If the scaphoid drops until it finds a natural resting-place on the floor in walking, what occurs to the astragalus and os calcis? These two bones are practically one, and unless the ligamentous connection between them is disturbed the os calcis must go up as the astragalus moves down. The shortened gastrocnemius gets shorter, the plantar fascia becomes stretched to its fullest extent, until finally we have a fully developed flat-foot.

If there were normal antero-posterior motion at the ankle center, the plantar tissues could not suffer. With normal motion here the weight of the body would be diffused about the astragalar curve—not lost on the more remote tissues at the sole of the foot. If there were a joint at the transverse axis of the ankle, as there is at the medio-tarsal transverse axis, we might have a yielding at the ankle center of motion. But while the astragalus moves downward, and the os calcis moves upward, the reverse movement being limited by the shortened post-tibial group, there is no transverse joint in either the os calcis or astragalus, and motion here occurs upon a remote arc (C D, Fig. 2).

It would seem comparatively easy to demonstrate flexion of the ankle in flat-foot. But it is not so easy as it seems. In making the examination and in applying tests it must be borne in mind that the usual bases of measurement are no longer present. The medio-tarsal joint is lowered, the plantar tissues are elongated, the "ball of the foot" is gone, and the inner border of the foot is lengthened. There is a greatly increased movement at the medio-tarsal joint, and the anterior part of the tarsus is abducted. It is impossible to restore these parts to their normal relation to make a normal test. Even if the anterior part of the foot seems to pass a point that indicates an approximately normal flexion, the heel does not come down with the movement upon the ankle center. Vicarious movement occurs at the medio-tarsal joint.

In the early stages of the condition the abduction may be easily overcome, because then there is no reflex spasm of the peronei. When there is reflex spasm present, it is impossible to place the foot in a position to test flexion of the ankle.

The only way to approximate the normal condition is to first overcome the abduction of the anterior part of the tarsus. Then attempt to restore the arch by manual pressure and support with a relaxed gastrocnemius. See that the knee is in full extension and that the entire tarsus falls directly under and in normal line with the long diameter of the tibia. Then gradually and slowly flex the ankle, making the pressure as near the medio-tarsal joint as possible, so as to avoid using the vicarious medio-tarsal motion; and, finally, having done all this, use the outer border of the foot as a plane of measurement. Bear in mind that we are testing antero-posterior motion at the ankle, not at the medio-tarsal joint.

If these precautions are properly applied, it will be seen that very imperfect flexion occurs at the ankle center of motion; and even if an apparently slight flexion is found, it does not follow that it is a true flexion at the ankle.

The os calcis can not be brought down, and the altered relation of the tarsal bones to each other makes an artificial rather than a real flexion.

I do not pretend to say that a shortened gastrocnemius is the only cause of flat-foot, any more than I would claim that tuberculosis was the only cause of chronic inflammation at the hip-joint. But I do say that it is a very frequent, and indeed the most frequent, cause of flat-foot, and, so far as I know, it is a hitherto unrecognized cause. And I may even say that among the hundreds of cases that I have examined during the past eight or ten years, I do not recall over three or four instances where I could not satisfy myself that the tendo Achillis was tense and unyielding when flexion was attempted beyond ninety degrees. Frequently it would be something more than ninety degrees, and in the exceptional cases referred to flexion would stop at little less than ninety degrees—say, eighty-nine or eighty-eight degrees—but always far less than true normal flexion. If flexion stops at ninety-five degrees, I should not expect to find flat-foot. I would rather expect to find shortened plantar tissues with adduction of the foot. I have followed a large number of flat-foot cases, as we all do in dispensary work, and I am convinced that the peroneal shortening is secondary to the gastrocnemius shortening; and I am satisfied from clinical observation that the spasm of the peronei is due to an osteitis located in the tarsus and caused by prolonged traumatism.

One can easily imagine that a sufficient degree of direct traumatism may be inflicted upon the tarsus to cause a flat-foot, or that hereditary causes may predispose one to acquire it easily and from slight causes, or that a peculiar gait arising from causes operating from above—*i.e.*, knock-knee, etc.—might occasion flat-foot. All these causes, however, are exceptional. The flat-foot I am attempting to describe is the classical flat-foot, where none of the exceptional causes are present, and where almost uniformly the conditions I have attempted to describe are found.

The conditions in non-deforming club-foot vary from those found in flat-foot largely and almost only in the plantar and tarsal conditions. In non-deforming club-foot the plantar tissues may successfully resist the influences which produce a relaxation in the other class of cases. I have seen typical cases of non-deforming club-foot become typical cases of flat-foot. At twelve or thirteen, in young girls, the non-deforming club-foot (frequently associated with true lateral curvature and muscular eye troubles) was present. At sixteen or eighteen flat-foot had developed.

If we further study the various ankle-joint conditions we may find confirmation of the views here expressed. The retraction of the gastrocnemius muscle after Hey's or Chopart's operation, and, indeed, in any amputation which removes the foot anterior to the medio-tarsal joint, but which leaves the lateral muscles attached to the stump, may be taken as illustrations of the action of the post-tibial muscles in producing flat-foot. In ankylosis of the ankle-joint after chronic inflammation, where no motion occurs at the transverse axis of motion, flat-foot does not occur. Indeed, it can not occur. If all motion is prevented at the astragalar curve, it can not occur at the os calcis or at the medio-tarsal joint. The conditions in paralysis of the leg have already been referred to. Indeed, it is hard to imagine a condition of flat-foot with a paralyzed or elongated gastrocnemius, just as it is an easy matter to demonstrate that a shortened gastrocnemius is essential to its production.

And yet flat-foot is very common, much more so than is generally supposed. Many persons go through life with a slight form of it and complain a good deal and consult their shoemaker and chiropodist, but rarely go to their medical attendant for advice. And even if they do, the patient is very likely to be referred back to his shoemaker or an instrument-maker, who fails either to recognize the trouble or to relieve it.

The cure of flat-foot would be easily accomplished if one could temporarily paralyze all the extensors of the ankle-joint, and wait for the slow process of plantar tissue shortening that occurs when the gastrocnemius is paralyzed in poliomyelitis anterior.

This, of course, is impossible. But we may imitate Nature and follow her methods as closely as possible. We might divide the tendo Achillis, as we would in a more marked case of equinus, and, if necessary, supplement this by the division of the peronei and tibialis anticus. This would enable one to crowd the os calcis forward and thus raise the depressed astragalar neck. The plantar tissues would relax under this procedure, and the parts could be approximately restored to their normal position, and held there by apparatus that would adduct the foot, thus imitating the action of the tibialis posticus. This adduction of the foot should be kept up for a period of weeks in the recumbent position and then followed by the use of an ankle support that would accomplish the same end.

Or we might stretch forcibly, under ether, the gastrocnemius and the peronei, and overcome the deformity even to the extent of supercorrection (an operation which I performed as long ago as in 1873) and maintain adduction for as long a period as necessary in apparatus. It is apparent to me that in practising this procedure I have unwittingly elongated the gastrocnemius in my efforts to overcome the more feeble extensor contraction at the peronei. Certainly in old and confirmed cases this would seem to be the better plan, especially among the poorer classes, where time is of value to the patient—although I have successfully relieved quite a number of adult and confirmed cases by a simpler and, it seems to me, more rational proceeding, which does not require etherization, and which permits the patient to pursue his daily occupation.

The first point of attack is the shortened gastrocnemius—for whether it is admitted or not that this muscle is shortened, all will agree, I think, that it will do no harm to bring the heel downward and forward toward the distal end of the elongated plantar tissues.

The original traction-shoe stretches the plantar tissues as well as the gastrocnemius. This, of course, contraindicates its use in flat-foot. Recent experiments with it have enabled me to obviate the plantar-fascia stretching, and I have obtained many excellent results by its use.

One essential modification consists of a curved plate made to fit the normal plantar arch. This plate slides backward and forward upon the foot-piece of the apparatus, and can be adjusted to any desired position. The other modification consists of a transverse worm and screw placed in the traction rod and just anterior to the point where it leaves its sheath. By using this worm and screw the anterior part of the foot-plate can be thrown downward away from the tarsus and metatarsus, while the part immediately under the medio-tarsal joint is pushed up. By using the key which brings the heel part of the apparatus downward and forward, and then applying the upward pressure of the plantar plate, the heel is drawn downward and forward and the neck of the astragalus is thrown upward and backward, following the curved lines

B L A and F K E in Diagram No. 3. By this apparatus a firm hold is obtained upon the os calcis and astragalus.

And so every day, for a few moments, the os calcis is pulled downward and forward, and the astragalus is pushed upward and backward, and after using the stretcher an ankle support is applied which causes the patient to walk on the outer border of the foot, not by building up the sole of the shoe, for this is a fallacy, but by so constructing the ankle piece of the ankle support that it is lower on the outside than on the inside. This forms an unyielding support which does not adapt itself to the outline of the foot as any leather shoe will sooner or later do.

At night a simple apparatus is worn which holds the heel down, and which also adducts the foot, so that both night and day the plantar tissues are receiving physiological rest.

The ankle support has been used quite extensively in the New York Orthopædic Dispensary and Hospital without the stretcher, and many good results as to relief from pain, ability to walk and work, are reported.

PART IV

Miscellaneous

ON THE MECHANICAL TREATMENT OF UNUNITED FRACTURE OF THE NECK OF THE FEMUR, WITH TRACTION APPARATUS PRODUCING ABDUCTION OF THE THIGH, AND DIRECT LATERAL PRESSURE OVER THE TROCHANTER MAJOR[1]

Read before the American Orthopædic Association, in Washington, D. C., May 4, 1897.

On the 16th of December, 1885, I was asked to examine Mr. M. P. G., aged forty-two years, who gave the following history:

Case I.—On the 1st of October, 1885, Mr. G. was thrown from his horse and sustained an intracapsular fracture of the neck of the femur on the left side. The accident occurred a few miles from New York, and the patient was at once brought to his city residence, where he was treated by the "Buck's extension" method under the most favorable conditions by one of our best-known surgeons. At the end of ten weeks an examination proved that the fracture had failed to unite and the patient was given to understand that he must expect to be lame all his life—the alternative of a surgical operation being positively declined by him. I had known the patient previous to the accident, having prescribed for a weak knee of the right leg. He sent for me to ask if anything could be done from an orthopædic standpoint. At first I was inclined to advise the surgical procedure, but the patient declined to entertain any proposition of this nature. He was possessed of an indomitable will, and he said he would undertake anything which I suggested which would give a chance for recovery, or even relief.

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[1] *The New York Medical Journal*, for October 23, 1897.

Three cases of ununited fracture of the shaft of the femur—two at about the middle of the femur and one just below the trochanter minor—had come under my observation and treatment, and had been successfully treated by the long traction (hip) splint and an accurately fitted coaptation splint; and two cases of unilateral congenital dislocation of the hip-joint had been very much benefited by the same method of treatment; the coaptation splint in the latter cases being a firm belt properly padded, passing around the pelvis, making a direct lateral pressure over the trochanter major. In all these cases the limb had been restored to approximately its normal length by the traction splint, and this length had been maintained for a sufficiently long period. Complete bony union had resulted in the fracture cases, and an improved position with a firm artificial joint had resulted in the dislocation cases. I had long felt that the treatment of recent fracture of the neck of the femur lacked especially a sufficient means to control the fragments and keep them in apposition; but no opportunity to treat any recent cases of this nature had presented itself in my practice.

I explained to Mr. G. the method by which we might hope to secure a union of the fracture, based upon the results just related, but I also informed him that it would be an experiment and that I would not undertake it unless the plan was approved by a formal consultation. The late Dr. Henry B. Sands, who had been familiar with my ununited fracture and congenital dislocation cases, was consulted, and after examining Mr. G. he gave the proposed treatment his fullest sanction.

The thigh and leg were in the characteristic position of fracture of the neck. There had apparently been no attempt at union; a flail-like movement was present at the point of fracture and the limb was two inches and three quarters shorter than its mate. The patient was in excellent health and spirits, and the experimental treatment was commenced under favorable auspices.

On Christmas day, 1885, the long, straight, Taylor hip splint was applied. This was almost to a day twelve weeks after the original injury. In four days the limbs were of equal length. A belt, about three inches and a half wide, made of surcingle material, such as is used by saddlers, was now passed around the pelvis, a crescentic-shaped horsehair pad being placed over the trochanter major. This belt was firmly buckled at the opposite side of the pelvis. The limb was now placed in abduction at an angle of about twenty degrees, the origin of the adductor muscles being used as a fixed point to throw the distal toward the proximal fragment. A lever was thus created where the fulcrum (the origin of the adductors) was between the power (the lower end of the limb) and the resistance (the seat of the fracture). The entire limb was now placed on an inclined plane at an angle of about a hundred and thirty-five degrees.

I felt very sure, as I studied the conditions from day to day, that the traction maintained the length of the leg, and that the abduction of the thigh approximated the fragments. But I did not feel certain that my "surcingle" was making a sufficient lateral pressure. I felt that the "surcingle" was the weak point of my apparatus. I then further increased the lateral pressure by passing a tourniquet over the padded surcingle, and after that I had at hand the means of absolutely controlling the lateral pressure. When the patient was moved in bed for any purpose, or when the traction was modified in any way, or when the perineal pads were loosened, the tourniquet pressure was carried to the point of toleration. At other times the tourniquet pressure was modified.

This pressure did not give rise to any trouble, nor did it seriously interfere with the circulation.

Among the objections urged by some of my critics and friends in Mr. G.'s case was one which especially appealed to me. The knee-joint had already been immobilized about twelve weeks, and it was justly feared, if the fixation of this joint was prolonged during a further period of, perhaps, months, that it might become seriously damaged. Before applying the straight traction splint, I had therefore carefully measured the patient for an apparatus which combines all the advantages of the straight traction splint with an arrangement at the knee by which motion could be secured at this articulation whenever necessary.

On January 3, 1886, I applied this splint. I will briefly describe it.[1] It has the conventional pelvic band, which is strong and solid, with the ordinary perineal pads. It has an abduction screw, by which the shaft of the apparatus can be placed in any desired lateral position, and it has a mechanism at the knee by which motion can be made whenever desired at this joint. It also has two traction rods, one at the thigh part and one at the leg part, each with separate adhesive-plaster attachments. By this arrangement, traction can be made upon the thigh, while that at the leg is removed or modified. During the treatment, after the first two weeks, the patient was drawn down to the foot of the bed, until the knee-joint was opposite this point. The leg traction was then modified, and the thigh traction increased. We were thus able to flex the leg without disturbing the thigh traction. While this was being executed, the tourniquet pressure was greatly increased. The knee was thus "exercised" every second day without trouble and without pain and without any detriment to the fracture.

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[1] Further experience proves that this complicated form of apparatus is not necessary. A simple, long traction hip splint, based upon the Davis-Taylor principle, and which can be obtained from any instrument maker, answers every indication in both acute and chronic cases.

On the 19th of January, 1886, Mr. G. was successfully removed to Lakewood, New Jersey. On the 28th of January he was placed on his feet with the use of crutches, a high shoe (two inches) being placed under the foot of the sound limb. He commenced at this time to walk on crutches, until he daily walked half a mile or more out of doors. He was allowed to stand up or to walk at will, but he was never permitted to sit down, or to try to sit down. His health remained good in every way, and on April 11th, about four months after treatment was commenced, Dr. Sands saw the patient with me and found a solid bony union. It was deemed best, however, not to discontinue the protection of the traction apparatus. A modified (perineal support) treatment was kept up until June 5th, when he was discharged cured.

At the final examination the limb was found to be three fourths of an inch shorter than its mate. The patient could walk well. He did not need a high sole to compensate for the difference in the length of his legs. All the motions of the hip were somewhat modified. Flexion was almost complete; abduction and adduction were slightly less than normal; extension of the thigh was resisted somewhat, while rotation in and rotation out were somewhat limited.

I saw Mr. G. in London last summer. No one would notice that he had anything more than a peculiar gait. He is not lame. He walks long distances, hunts and rides on horseback, plays tennis, etc., as well and as actively as if his hip had never been injured.

Case II.—On the 9th of October, 1889, I saw Miss X., aged forty years. Eighteen months previously she had received an injury in Europe, and had sustained a fracture of the neck of the femur. The limb was three fourths of an inch shorter than its mate and a distinct "telescoping" could be demonstrated, as well as a distinct crepitus, at the point of fracture. In consultation with Dr. Charles McBurney on October 15th the diagnosis of intracapsular fracture was confirmed. I submitted to Dr. McBurney a plan of treatment similar to that which had proved successful in Mr. G.'s case, and he expressed himself as fully approving the effort. The patient was informed that the issue would be doubtful, and that the length of time which had elapsed since the injury made the treatment an experiment. After due deliberation she decided to make the attempt.

It was decided to pursue the treatment for three months, and then, if there was no evidence of union, the effort was to be abandoned. Accordingly, on October 30th, the long traction splint was applied with the abduction screw, the surcingle, and the tourniquet, and the same treatment outlined in the previous case was very carefully carried out. The patient was seen by Dr. McBurney on November 15th, and again on December 6th. On February 1, 1890, three months after the first application of the traction splint, the apparatus was removed, and Dr. McBurney and myself made careful and thorough tests of the fracture. The telescoping had disappeared, and the crepitus could not be detected. There seemed to be a pretty firm union of some sort, though whether it was ligamentous or osseous we could not decide. The patient could not raise the limb from the couch with the knee extended, though she could rotate it through quite a considerable arc. It seemed that there was enough to encourage us to make a further effort. Before doing anything further, however, we decided to remove the apparatus for a couple of weeks to see if any shortening took place. After nineteen days no shortening had occurred. The traction apparatus had brought the limb down half an inch, or within a quarter of an inch of its normal length, and for nearly three weeks, without any apparatus, except the surcingle during the day, this length had been maintained. The patient went about on crutches. During the interval, Dr. W. T. Bull also saw the patient and examined the conditions present. Both he and Dr. McBurney deemed the evidence, while not positively indicating bony union, as affording a hope that this result might occur. On February 25th the treatment was again commenced and it was steadily maintained until the 6th of May, when Dr. Bull again saw the patient.

The conditions again seemed favorable, but not positively indicating bony union. There was no telescoping; no crepitus; no shortening under severe manual tests. Indeed, there was some sort of union. It was hoped that it might prove to be bony union. Up to July 1st, when I went away on my vacation, the limb had not shortened. No direct evidence of fracture could be elicited, and yet there was an expressive disability about the limb which pointed to an uncertain result. It was proposed at this juncture to apply a perineal walking support, so as to maintain the advantage gained by the prolonged treatment, but the patient had had a previous very unpleasant experience with an apparatus of this kind, and positively refused to have one adjusted. After I went away the patient came under the care of Dr. Bull. The patient was instructed to use the limb with the protection of crutches and to gradually bring the weight of the body upon the fracture. During the summer, under these conditions, the old condition gradually returned. The telescoping reappeared, the crepitus came back, and the thigh slipped back to its old position of three fourths of an inch shortening. The result was a failure after all.

It would seem, however, even in this case, where treatment was commenced eighteen months after the fracture occurred, that Nature made an effort to secure union. It was a pretty firm union too, and one which, for a while, resisted all ordinary tests. It may even be that had the treatment been absolutely uninterrupted, and had it been persisted in for a longer period, with a perineal support after actual treatment was stopped, a serviceable union might have resulted.

I do not propose on this occasion to review the various methods which have been devised for the treatment of fracture of the neck of the femur. Both of these cases were ununited fractures and they came under my care because the conventional method had failed. The means used were the result of some thought and study upon the subject, and when I treated Mr. G. there was nothing that I could find in medical literature to guide or aid me.

Since my first case, Dr. Nicholas Senn has published an able essay on The Treatment of Fracture of the Neck of the Femur by Immediate Reduction and Permanent Fixation. This essay is published in the *Journal of the American Medical Association* for August 13, 1889, and it presents a comprehensive review of the subject, and the author reaches conclusions which I think the medical profession must approve as applied to recent cases of this class of fractures.

Dr. Senn depends, however, upon manual traction for the first reduction of the fracture, and then adds a comprehensive gypsum splint with a screw pressure over the trochanter major. No attempt is made to throw the fractured ends together by abducting the limb, and he depends upon the fixing power of the plaster-of-Paris splint to maintain the traction induced by the first manual effort.

What I deem to be the essential element in the treatment, both of cases of recent and ununited fracture, could not be successfully carried out by the use of the gypsum splint alone, viz., the permanent abduction of the thigh. The gypsum splint would not have sufficient resistance to hold the limb against the unremitting action of the adductor muscles. A constant and easily adjusted form of traction is needed to secure this result, and if this traction is successfully kept up the lateral pressure upon the trochanter may be very materially reduced.

A few words in closing: My experience leads me to say that the tourniquet pressure over the trochanter major will not produce any excoriation or any serious interference with the circulation. In neither of my cases did it produce anything more than inconvenience. Indeed, I was surprised to find how little pressure was needed to maintain the fragments in apposition with the conjoined rack and pinion traction and abduction. Still further: The long traction splint (Taylor's), such as is used in the treatment of hip-joint disease, with an abduction screw at the junction of the traction rod with the pelvic band, and with the surcingle and tourniquet, makes a complete apparatus for the treatment of recent cases of intracapsular fracture. It meets all the indications, and certainly, if good results can be obtained in cases of non-union of long duration, its use is strongly indicated in acute cases. The ease with which the patient can be placed upon a bedpan, etc., the certainty that the fractured ends can always be kept in apposition, the readiness with which the patient adapts himself to the treatment, the rapidity with which he gains confidence and walks about with the high shoe and crutches, and finally the absence of a complicated technique, all commend this method.

The above remarks were submitted to the Surgical Section of the New York Academy of Medicine on June 13, 1892. Since that time the following cases have occurred and are herewith appended to this report. The paper was not published for the reason that it was deemed best to secure additional evidence before finally presenting the matter to the medical profession.

Case III.—Reported by Dr. T. Halsted Myers. J. W. D., aged forty-two years. While driving, the patient was thrown from his carriage and was stepped upon by the horse. Inability to stand and great pain were immediately observed. Nine weeks after the accident, on September 24, 1894, Dr. Myers found five eighths of an inch shortening, bony crepitus, "telescoping" at the neck of the femur, and the characteristic position of fracture at the neck of the femur. On October 3, 1894, the long traction splint was applied and the patient, who was obliged to support himself, went about on crutches, with a high shoe on the sound limb. A pelvic girdle was also applied soon after the treatment was commenced. On January 4, 1895, all pain had ceased and the telescoping and crepitus had disappeared. Although still advised to keep quiet, the patient insisted upon going about on crutches. On January 25, 1895, a "short splint" was applied, the crutches and circingle being continued. On May 10, 1895, the evidences of bony union being positive, all apparatus was removed. The patient had no pain and walked well.

At an examination made February 21, 1896, perfectly firm bony union was found with excellent motion at the hip-joint. Patient rides horseback, etc., with ease. Shortening, half an inch.

Case IV.—C. M., aged twenty-two years, a Norwegian sailor, applied at the New York Orthopædic Dispensary and Hospital on November 16, 1894, with the following history: On May 24, 1894, he fell several feet aboard ship, injuring his left hip. He was treated in a general hospital in this city for five weeks, with a plaster-of-Paris splint, the patient says, for dislocation of the hip-joint. After five weeks he was discharged, walking upon crutches. There was no further treatment, and the condition not improving the patient applied to the dispensary, as above stated.

On examination there was found an inch and a quarter shortening of the affected limb, a distinct crepitation, nearly an inch of "telescoping," and the characteristic position of intracapsular fracture of the neck of the femur. This diagnosis was made and confirmed at subsequent examinations.

As the patient had no home and was living in a sailors' boarding house it seemed useless to attempt to commence treatment. After a time I succeeded in obtaining permission to have the patient enter the Presbyterian Hospital, where, through the courtesy of Dr. Andrew J. McCosh, attending surgeon, the writer was permitted to give the patient his personal attention.

On January 21, 1895, nine months after the accident, with the assistance of the house staff of the hospital and Dr. Hibbs, house surgeon to the Orthopædic Hospital, a long traction hip splint was applied, the surcingle belt was adjusted, the thigh abducted, by making the distal perineal pad of the apparatus tight before making the traction, and the limb placed on an inclined plane at about a hundred and thirty-five degrees. At this time careful measurement showed an inch and a half of shortening.

Dr. Hibbs assumed the personal care of the patient and saw him regularly.

On May 23, 1895, I examined the patient and found evidence of bony union. It was advised that the same treatment be continued, however, as it did not seem positive that the union was complete.

On July 30, 1895, bony union was complete; the patient could raise the foot from the bed with the knee fully extended. The use of the apparatus was continued, and the patient was directed to get up and move about with crutches and a high sole to the unaffected limb. There was three fourths of an inch shortening.

As the patient had no home he was kept in the hospital by the kind permission of the authorities until December 17, 1895, when he was discharged well, with three fourths of an inch shortening.

On November 17, 1896, when I saw the patient last, he still had three fourths of an inch shortening. At that time his gait was good and he had been at work for some months. The bony union was very firm, and every function of the joint was well performed, except that there was some limitation of motion at the hip in all directions. The knee-joint was about normal in its movements.

Other cases of impacted fracture accompanied by pain and great disability have been treated by this method with much success. Dr. Myers reports two and the writer has had three, in all of which rapid improvement followed the use of the traction splint in a few weeks or months.

In all these cases crutches with a high sole were used during locomotion.

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COMMENT BY

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June 21, 1923.

Dr. Shaffer's paper on "Mechanical Treatment of Ununited Fracture of the Neck of the Femur" came to my attention some years ago and awakened my interest because of the clearness of recognition of the mechanical factors and the efficiency with which treatment seemed to have been carried out in the few cases listed.

We have heard so much of late about abduction treatment of hip fractures and so much of which has been predicated on the basis of mechanics which does not seem to be reasonable, that it is very interesting to read Dr. Shaffer's note "The limb was now placed in abduction at an angle of about twenty degrees, the *origin of the adductor muscles* being used as a *fixed point* to throw the distal toward the proximal fragment. A lever was thus created where the fulcrum (the origin of the adductors) was between the power (the lower end of the limb) and the resistance (the seat of the fracture)."

Moreover the necessity of preliminary traction to bring the surfaces opposite one another, the usefulness of pressure over the trochanter, all were clearly recognized. Moreover, the use of a splint instead of plaster, more accurate surely, may give better results than our more recent routine, which can not yet be regarded as fully efficient in these cases of loose hip fracture.

NOTE BY THE AUTHOR

(September, 1923.)

Confronted, in 1885, by an entirely new problem in the treatment of fracture of the neck of the femur, without any precedent to follow, or authority to consult, the author feels that his success in the treatment of the cases of this fracture, recorded above, deserves, at least the credit of being the first application of the underlying principle of abduction of the thigh in the treatment of this condition.

Dr. Cotton calls attention to the method I originally used.

In devising the treatment I realized that the proximal fragment—the detached head of the femur, was not unlike a billiard ball lying in a tea cup, easily susceptible to displacement upon the application of a slight force, and that we were without any means of splinting it except by the distensible capsule. I felt that over abduction was to be avoided. In carrying out this idea I made the tension of the adductor tendons my guide, having at hand a certain method of controlling the amount of abduction—a point that was daily watched. I likewise had an available and easily controlled traction force. The length of the leg was always maintained. These two factors, combined with a pressure over the trochanter, that could be regulated at will, contributed to the good results obtained.

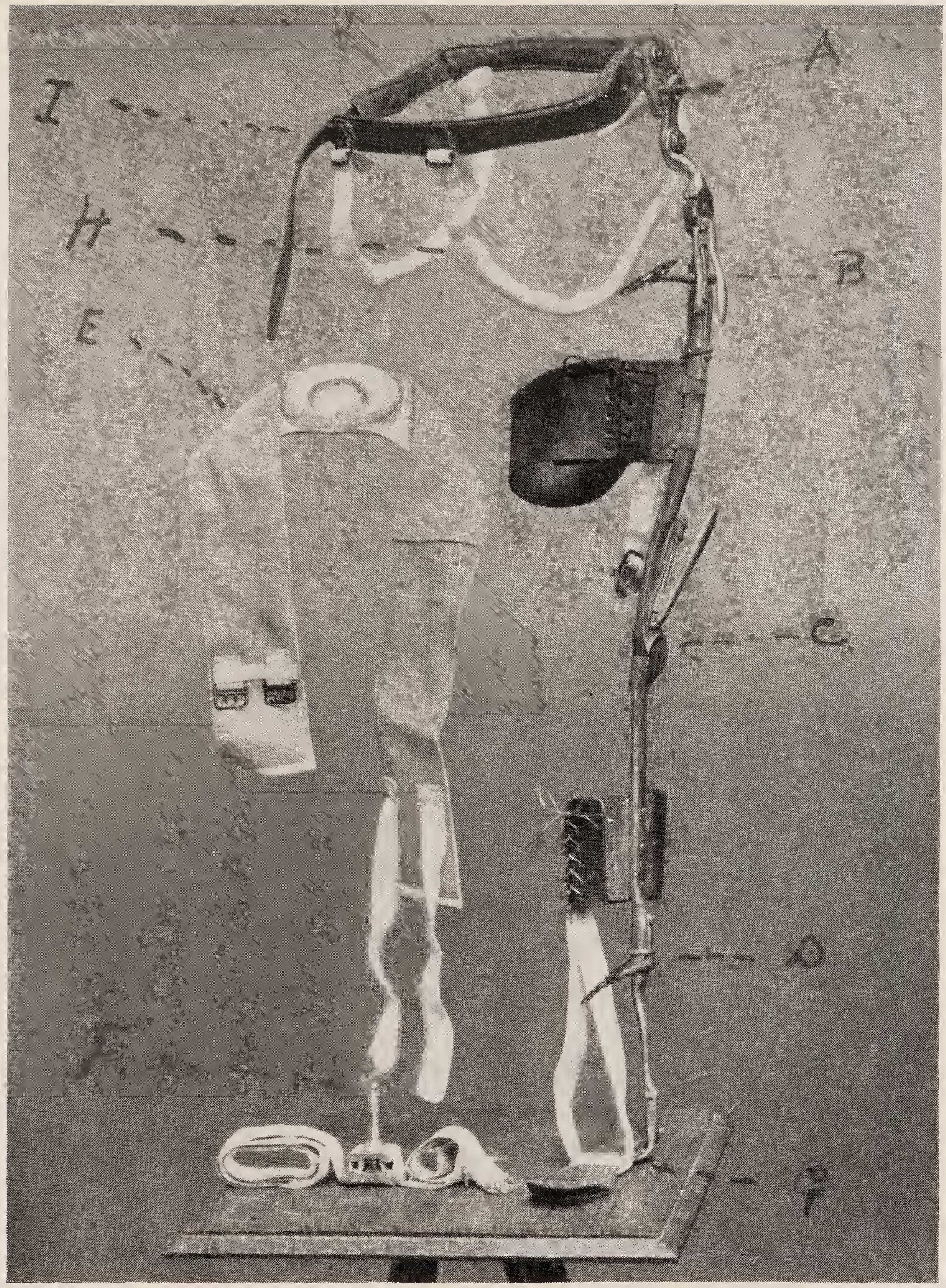
The one thing necessary, in the successful treatment of any fracture, is the accurate coaptation of the fragments. I do not see how this can be accomplished with any degree of certainty, in fracture of the femoral neck by the necessarily haphazard application of the gypsum splint to the abducted thigh, especially without a direct and continuous direct linear traction—and many of us know that continuous traction cannot be maintained by the plaster-of-Paris bandage.

I append a reproduction of the apparatus I used. It is a traction splint of my own device. (See illustration.) This specially devised splint was adjusted about three weeks after the simpler long splint was applied. The success of the treatment was undoubtedly made more certain by its application and use.

A, represents the abduction screw; B, the thigh traction rod; C, joint at the knee, controlled by a lever; D, the leg traction rod; G, the firmly fixed foot-plate over which are passed the webbing straps, which are attached to adhesive strips encircling the leg and thigh, by which traction is made. (This plate can be worn inside the shoe): E the surcingle belt, with the horse shoe pad, to pass around the pelvis over the trochanters; F, the tourniquet by which any amount of pressure can be produced; H, the perineal pads; I, the firm steel hip band. The leg and thigh leather bands need no explanation.

I think it well to reproduce this apparatus in detail, as some think that I used a simple long Taylor hip splint during the entire treatment. I devised this apparatus to meet all the indications, after an exhaustive study of the conditions.

I stated in my paper that "a simple long traction hip splint . . . answers every indication in both acute and chronic cases." Experience proves this to be an error and I would modify this statement by saying that the abduction screw and the surcingle should be added. Direct linear traction could thus be applied, the required amount of abduction, under supervision, could be secured, and the pressure over the trochanter maintained, with or without the tourniquet.



Apparatus for fracture of the neck of the femur

ON ELONGATION OF THE LIGAMENTUM PATELLÆ AS A FACTOR IN THE PRODUCTION OF CERTAIN KNEE TROUBLES AND DIFFICULTIES OF LOCOMOTION [1]

In the year 1873, a man of about twenty-five years of age applied at the New York Orthopædic Dispensary, complaining of considerable difficulty of locomotion, and especially of great difficulty when going up and down stairs. The patient was supposed, by the physician who referred the patient to the Dispensary, to have an insidious form of paralysis.

Various tests and a careful examination proved that there was no paralysis present. The patient then said his knees were very weak, and an examination of these joints showed a very peculiar state of affairs. The patellæ, when the patient was in a sitting position with the knees flexed just beyond 90°, were drawn above the condyles of the femur, and were lying in a plane almost horizontal with the long axis of the femur. The ligamentum patellæ on either side measured three and a half inches long and the altered joint outline was characteristic of these peculiar conditions. The patient stated that he had to walk very carefully, or he would, upon the slightest lateral jar, dislocate one of his knee-caps outward, and fall suddenly to the ground. There was a slight knock-knee on either side, and there was present at the intercondyloid notch a very perceptible tumor—an apparent exostosis—which occupied the position at which the patella would normally rest.

Several attempts were made to relieve the condition by the use of apparatus. He finally obtained some comfort and a little relief by using a silk elastic knee-cap with a lateral pad crescentic in shape, and made of rubber, which prevented the outward dislocation of the patella. The patient was lost sight of. He knew no cause for his trouble.

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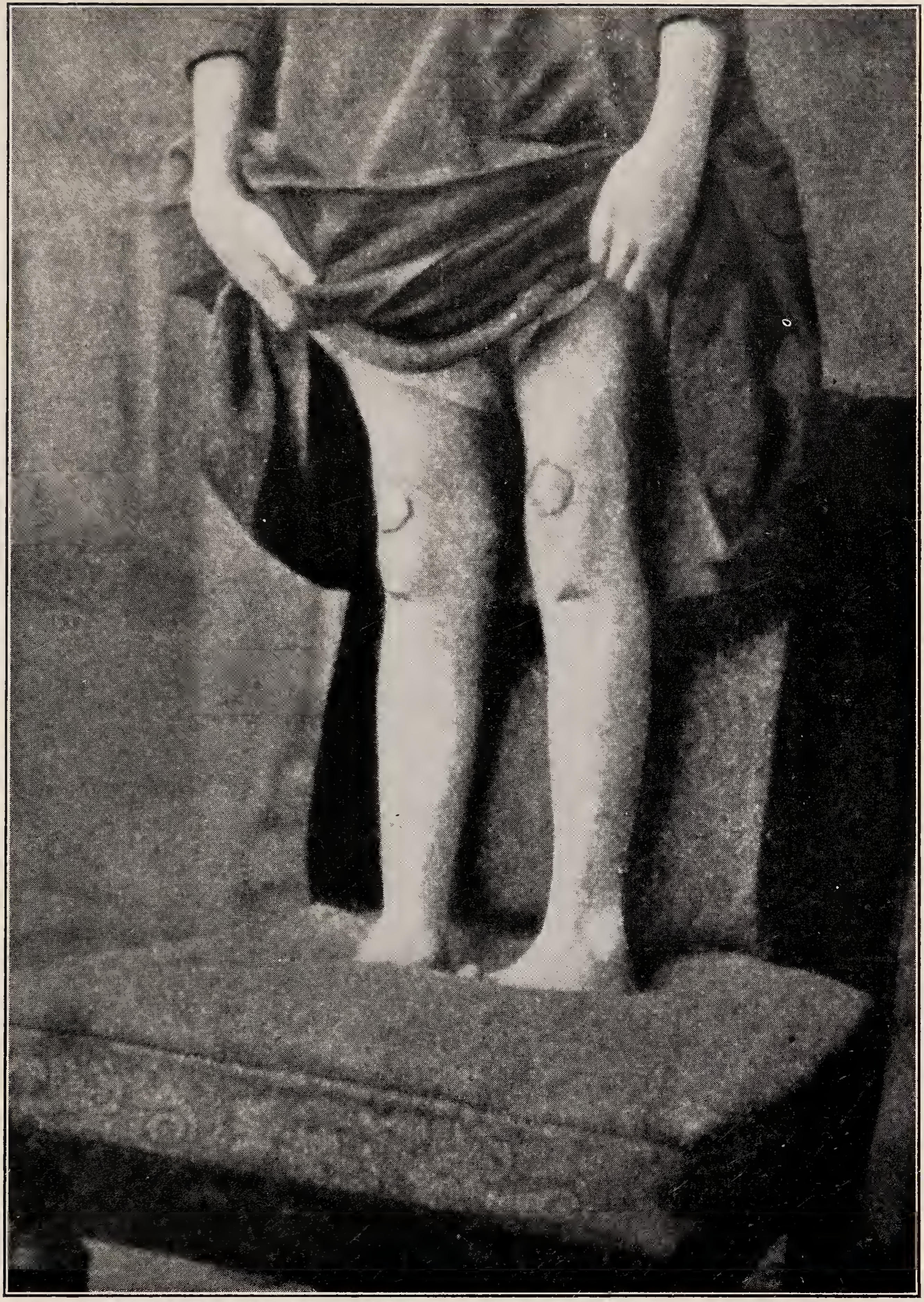
[1] *Transactions of the American Orthopædic Association*, Sept., 1891.

This case made an impression upon me, and a photograph was taken at the time, but it has become lost or mislaid in the years that have elapsed. It was not until a later period, when an opportunity occurred to watch the progress of a similar condition, that this case became of value.

In 1884, I was called by Dr. Janvrin to see a young lad of fourteen years who had hip-joint disease. The usual traction apparatus was applied, and, at the end of two years, the hip trouble was entirely removed. About this time, however, the patient began to grow very rapidly and to show signs of a progressive knock-knee. The member (right) affected with hip-joint disease was quite straight, but the opposite side showed a very considerable genu valgum.

An examination of the ligamentum patellæ showed that it was three and one fourth inches long on the left side; on the opposite side it was three inches long. The evident upward displacement of the patellæ on either side was marked during flexion of the knee at right angles. There was a distinct tumor in the intercondyloid notch on either side, more marked on the left; and the patient moved up and down stairs with an awkward gait and fell at times without apparent cause. During his first summer in the country, after the hip disease was cured, he fell and dislocated his right patella. This was reduced by the family physician. The following year, he dislocated the left patella. In each instance, the fall occurred while the patient was walking on an irregular surface.

The dislocation of the left patella proved a serious affair. The surgeon in attendance failed to reduce it, and, when the patient returned to the city, all attempts to reduce it seemed useless. When replaced, the bone immediately slipped back into its old position. At a consultation, at which the late Dr. H. B. Sands, Dr. Janvrin, and myself were present, it was decided to make no further attempt at reduction, for the reason that the intercondy-



Elongated Ligamentum Patellæ

loid tumor and the elongated ligamentum patellæ would prevent the patella from moving in its true longitudinal plane. For the same reason, it was decided not to perform Macewen's operation, though the knock-knee was very considerable. Dr. Sands considered these reasons to be of sufficient importance to contra-indicate surgical interference.

With the use of my knock-knee apparatus, the genu valgum was much reduced, and the patient now has a slight knock-knee, a dislocated patella on the left side, and a very serviceable knee on the right side. His left ligamentum patellæ is still three and one fourth inches long; his right, three inches long. He can place his left knee in full extension, with the quadriceps extensor femoris acting upon the dislocated patella, which moves back and forth above, and to the outside of the outer condyle of the femur. When sitting with knee flexed, patient abducts and rotates thigh outward to secure the quadriceps action upon the patella. He flexes the knee by a semi-rotating movement. The tibia is rotated permanently outward, so that the tubercle of the tibia is fully one inch to the outside of its normal position. Even if the patella was in its normal position its line of movement, taking the insertion of the ligamentum patellæ as a guide, would be directly through the outer condyle.

By careful treatment, and especially by avoiding traumatism, and by the use of a firm conventional leg-brace, which gives lateral support and prevents the rotating movement of the tibia upon the femur, the patient now walks from ten to fifteen miles a day.

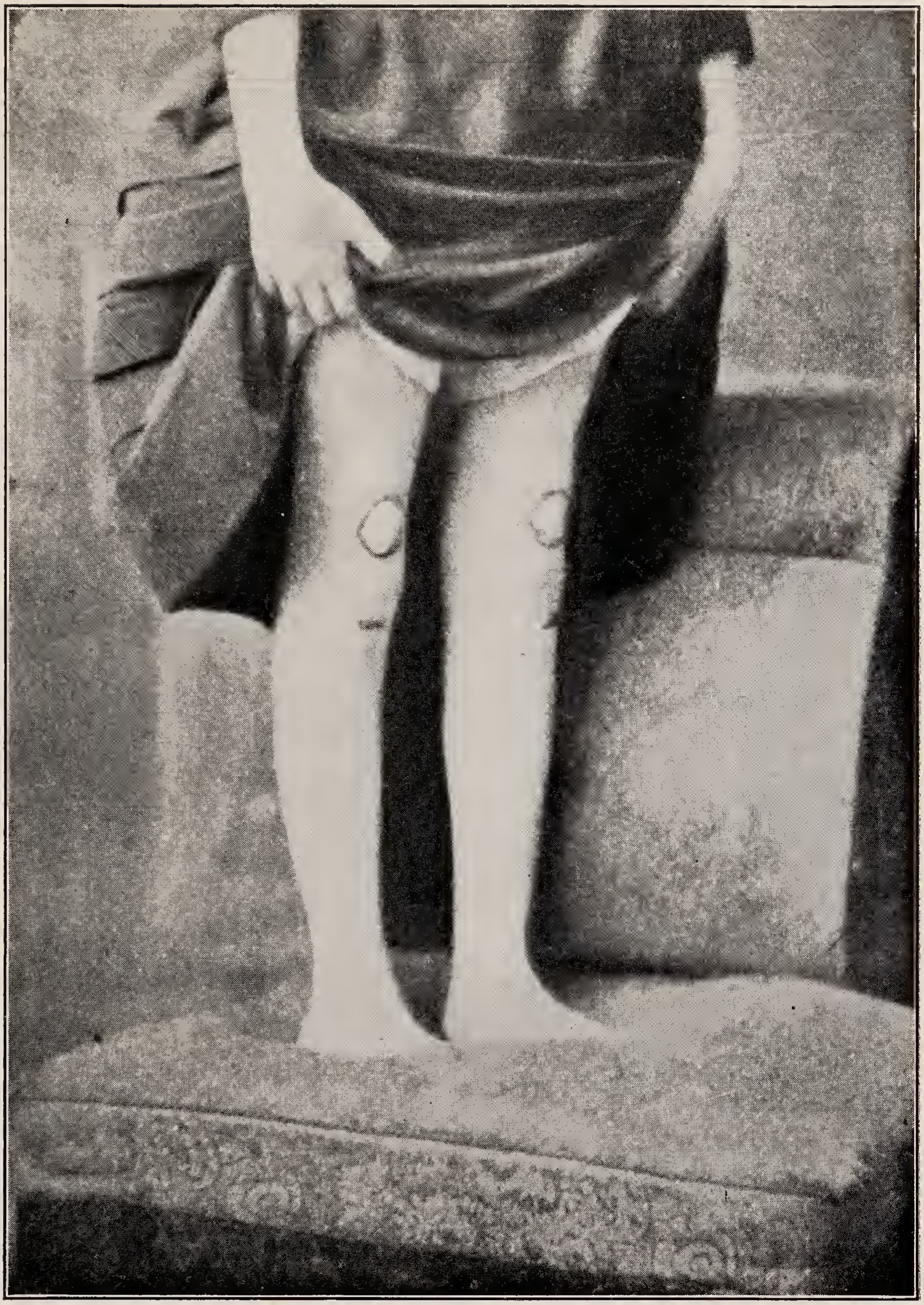
I next saw a lady, Mrs. E., aged about thirty-five years. She came to me, unable to walk, except with great difficulty, and always with danger of falling. The least irregularity of the pavement would cause a fall. She had never dislocated her patellæ. There was slight knock-knee on either side. The ligamentum patellæ on either side measured three inches, and the patellæ occupied the position indicated in the accompanying photographs of Maggie M. This patient had great difficulty in going up or down stairs, and the disability was almost identical with that produced by a transverse fracture of the patella, with a long ligamentous union. Light lateral "leg-braces," preventing lateral movement at the knee and arresting the rotating movement of the tibia during flexion, gave this lady almost complete relief. She could walk with great comfort, and without any fear of falling.

The following history (condensed) is taken from the books of the New York Orthopædic Dispensary and Hospital:

Maggie M., aged thirteen years. Admitted December 2, 1886. Two years ago fell on an orange-peel. Up to that time seemed all right. Since then falls easily and frequently. When she first fell, she dislocated her left knee-cap, which was reduced by a woman who saw the child fall. Right patella dislocated about two months ago by a similar accident. After each fall, the knee swelled, and there was some pain, which gradually disappeared. Previous treatment, "knee-cap," which did no good. Both limbs measure the same in length and circumference being about normal.

No knock-knee; very slight lateral mobility at knee-joint. Patellæ when knees are at rest in full extension lie above condyles and look obliquely upward and out. Patellæ freely movable, and, when relieved of muscular action, they can easily be brought down to normal position. Disability in walking, etc., exactly like that produced by fracture of the patella with long ligamentous union. There is an evident exostosis at sulcus between condyles of femur. Length of left ligamentum patellæ two and three fourths inches; right, two and five eighths. The exact condition and relation of the parts is shown in the accompanying photographs, the tubercle of the tibia and the patella on either side being outlined by ink before the photograph was taken.

The treatment consisted in the use of leg-braces limiting the lateral and rotating movements of the knee. The patient obtained some relief, but was lost sight of after a few months.



Elongated Ligamentum Patellæ

In the case of Miss X., whose knees had been for a long time immobolized in plaster-of-Paris dressings, with a consequent fibrous ankylosis, the patellar ligament was stretched by the force required to break up the sub-patellar adhesions. The right ligamentum patellæ is now two and three fourths inches long; the left, two and a half inches long. An expressive disability exists in this case, occasioned partly by the reduced quadriceps power in knee use, and partly by a disagreeable "click," which occurs when the patella, especially on the right side, slides into its supra-condyloid position.

Several cases of elongated ligamentum patellæ have come under my observation since. I will briefly relate two:

A young man, Mr. W., consulted me because he had great difficulty in keeping up his part in playing lawn tennis. His knees, as he expressed it, "gave out very quickly." His patellar ligaments were nearly three inches long, and the disability was easily explained. He had considerable pain after prolonged use of his knees. Heat and swelling were sometimes present. He wore knee-caps without relief. He declined to submit to an operation which had for its object a shortening of the elongated ligament. He would not wear apparatus, and so long as he does not use his knees too much, he is now comparatively comfortable, though he has occasional attacks of pain and swelling after over-exertion.

A lady, Mrs. V., well and strong, quite muscular, and, as she says, "never sick," found difficulty in going up or down stairs. In this case, the elongation of the patellar ligaments was very marked. The cause being explained and the remedies proposed being rejected, nothing was done, though the patient's trouble seems to be increasing rather than otherwise.

I might relate other interesting cases where there was knee trouble due to the lengthened anterior patellar ligament. In some of them there was heat, pain, and swelling, due to overstrain, but no true joint inflammation. In others, a sense of weakness and debility. In others, tendency to dislocation of the patella outward. In all, however, there was a greater or lesser degree of disability under severe, or in some cases, ordinary use of the joint. It was especially noted when much strain was brought upon the articulation in the flexed position.

I have not succeeded in persuading any of my patients to have a section of the ligament removed. The operation seems both feasible and justifiable. Dr. Sands, in the case reported, declined, as before stated, to perform this operation, principally because of the apparent exostosis at the point where the patella glides between the condyles. This exostosis has existed in almost all my cases, and its cause is very obscure. It would seem to be a hypertrophy due to the lack of the normal pressure of the patella, which in its acquired position does not rest in the intercondyloid space.

I have measured many normal anterior ligaments of the knee since these cases came under my observation. Its normal length varies from about one half inch in children to two inches in adult life. These measurements were made from the apex of the tubercle of the tibia to the lower edge of the patella, with the quadriceps in full contraction, with the knee bent at 90°, thus making the ligament taut. In some adults, the ligament is only a little over one inch long; in others it is about one and a half; and in a few tall persons, it is about two inches long when measured as above. Anatomists vary very much as to the length of this structure. For example, Gray gives it as three inches; Ellis, as two inches; Morris, as two and a half inches on its inner border, three and a half on its outer border. But these measurements represent the ligament deprived of all contiguous tissue, and are made upon the cadaver. The ligament extends, for example, below the apex of the tubercle. Clinically, the measurements made by myself are correct, both as applied to the normal and elongated patellar ligament—the points of measurement being the same in either case.

Shortening of the ligamentum patellæ is also a condition that I have seen quite often. It has generally occurred as the result of a disused or paralyzed quadriceps extensor femoris muscle. In some cases, where the anterior thigh muscles are paralyzed, as in poliomyelitis anterior, the patella will rest against the tibia, and no trace of the ligament can be felt. The same may be said of certain cases of ankylosis of the knee, where the patella has been late in attaching itself to the subjacent bone. The ligament shortens under the influence of gravity and disuse, following a diseased or paralyzed quadriceps, and it becomes very short after a time.

My experience proves that this condition of elongated ligamentum patellæ is neither rare nor unique. As to its cause, I can find no satisfactory clue, except, of course, in cases like Miss X.'s, where force was used to dislodge the adherent patellæ. It is altogether likely that, in most cases, the condition is either congenital, or acquired early in life; and the fact that the majority of my cases were women or children, the former being ladies of leisure and refinement, without the history of an injury, would seem to eliminate traumatism as a cause.

A peculiar feature of these interesting cases is the intercondyloid growth, which in Dr. Janvrin's case gradually assumed the character of a considerable osseous tumor. As a mechanical factor, it prevents the normal movement of the patella over the trochlea, and it is a persistent barrier to a perfect restoration of the parts. In Dr. Janvrin's case, three separate operative procedures were indicated to restore the parts to anything like a normal relation. 1st. Macewen's supracondyloid operation to remove the knock-knee. 2dly. To shorten the ligamentum patellæ. 3dly. To remove the intercondyloid tumor. The result of the conservative treatment advised by Dr. Sands would seem to sustain his position that this complex operation was uncalled for.

My conclusions may be briefly stated:

1st. Elongation of the ligamentum patellæ may produce weak, painful, and apparently inflamed knee-joints.

2d. Many obscure knee-joint troubles, as well as impaired or difficult locomotion may be explained by this condition.

3d. Elongation of the ligamentum patellæ may produce a disability very like that produced by an elongated ligamentous union after transverse fracture of the patella.

And finally, elongation of the ligamentum patellæ may be, and very frequently is, produced by forcibly breaking up a fibrous ankylosis of the knee-joint.

ON THE CAUSE AND MECHANICAL TREATMENT OF SUBLUXATION OF THE SEMILUNAR CARTILAGE OF THE KNEE-JOINT[1]

Read before the Section on General Surgery of the College of Physicians of Philadelphia, March 11, 1898.

"HEY'S Internal Derangement of the Knee-Joint," occurring as it does both from trivial mishaps as well as from major injuries, and being a not infrequent accident, it seems strange that there should be any doubt as to its essential nature, especially so as Hey wrote his description of the trouble so long ago as 1803. Mr. J. F. Knott, writing in 1882, in the June number of the *Dublin Journal of Medical Science*, discusses the cause of the trouble somewhat at length, and he thinks that there is not so much a dislocation of the cartilage as that "the margin of the condyle is *jerked over* the internal semilunar cartilage." Writing in 1887, in the article "Movable Bodies in the Joints," in "Wood's Reference Hand-Book," Dr. Joseph D. Bryant says: "The nature of the derangement described by Hey is still an unsettled question." A further study of the subject in Allingham's treatise, "Internal Derangement of the Knee-Joint," published in Wood's "Medical and Surgical Monographs" in 1890, will, I think, dispel all reasonable doubt in the matter. It may be profitable for us, however, to inquire into its exact mode of production, but I fancy that the essential nature of the trouble is well understood.

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[1] *Annals of Surgery*, October, 1898.

From these and other sources, it may be assumed, I think, that the "internal derangement" described by Hey is occasioned by a varying degree of displacement; by even, in many cases, an imperceptible subluxation of one of the semilunar cartilages. In brief, a visible or manually demonstrated existence of the subluxation is not always necessary. The trouble is so far "internal" that in many cases the ordinary physical signs of a dislocation are not to be observed or felt. I have seen several cases where the trouble was diagnosticated as a "sprain," and I know of two instances where the physical signs of the trouble were so inconspicuous that a diagnosis of "an hysterical joint" was made.

A prolonged study of many cases convinces me that, aside from the generally accepted view that this subluxation occurs while the knee is flexed and the leg is rotated, there are other contributive causes which have not been carefully investigated. In short, that it is not the simple fact that rotation of the tibia occurs at the knee during flexion and extension of the joint, for these are normal movements; but rather that there is a delayed or hindered extension and rotation which permits this accident to occur. It would seem, from my studies, that this subluxation is not likely to occur, and I doubt if it ever does occur, except perhaps in cases of violent traumatism, while the quadriceps extensor muscle is relaxed.

Nor is the trouble ordinarily considered as one which belongs to orthopædic surgery. Especially since Allingham's time it has been regarded, I think, as being within the domain of the general surgeon, and so it is in its purely operative aspects. But, as with chronic diseases of the spine and joints, it has its conservative side, and if orthopædic surgeons can offer relief and cure by mechanical means, the general surgeon will, I am sure, welcome their efforts and aid them in their work.

For many years I had pursued the conventional treatment of Hey's joint,—that is, I regarded it as a traumatic, acute joint lesion, from which, as in other acute joint lesions, recovery might be expected with the use of rest, cold, counter-irritants, etc., in a few weeks. I tried to combat the persistent recurrence of the dislocation with knee-caps of various kinds and simple appliances, which, as it turned out, were useless forms of apparatus. When the trouble came back I again resorted to the usual treatment of acute synovial effusions, until Allingham's monograph appeared, and after this period I uniformly referred the persistently recurrent cases to some general surgeon or to some general hospital for operation.

It was a case of this nature—viz., a persistently recurrent trouble—which led me to study the conditions very carefully and critically, and, as the case is an interesting one, I will detail the history somewhat at length, using this case as a text for some comments.

Case I.—In June, 1891, I was consulted by a young man, Mr. J., aged twenty-two years, who three years previously had been a victim of subluxation of the internal semilunar cartilage of the right knee. The accident producing the trouble had been very simple. He was attempting to board a slowly moving horse-car, and had placed his right foot upon the step, when the car suddenly moved forward. A sudden effort to straighten the knee and to quickly jump on the car produced the subluxation. He was treated by rest and a gypsum bandage, and recovered from the acute symptoms in about four weeks. In various ways and under very simple conditions the accident recurred from time to time up to the day I saw him, when he was suffering from his last attack, produced in this instance by suddenly turning to confront a friend who attempted to play some minor practical joke upon him. After rest, etc., had resulted in a subsidence of the acute symptoms, and I had advised that he place himself under the care of a general surgeon for operation, he appealed to me, asking a further consideration of his case, and begged me to devise some means by which he might avoid the operation.

In looking over the notes of my previous cases I found eight in which the histories were tolerably complete. In these eight the following conditions were present: (1) A very considerable lateral mobility of the joint; (2) an elongated ligamentum patellæ, and (3) in six of the cases an inability to extend the knee completely.

I examined Mr. J. very carefully after considering his appeal for further conservative effort, and I found all these conditions present.

With the knee flexed at a right angle, the distance between the lower border of the patella and the tubercle of the tibia was two and a half inches. It was nearly two inches on the left side. The patella, instead of occupying the usual position in the trochlea with a normal anterior ligament to hold it accurately in its place, was displaced upward fully half an inch. The quick "air-brake" action of the patella on the knee-joint at a given angle, under the influence of the contraction of the quadriceps extensor muscle, is much modified under these circumstances. The clamping of the tibia against the condyles of the former is delayed, and the action of the patella becomes mechanically imperfect, the force being lost obliquely from before backward rather than against the condyles in a line more nearly in the long axis of the femur. The elongated anterior ligament also allows the lower border of the patella to tilt upward, away from the trochlea, as will be seen in the accompanying X-ray picture (Plate I, Case III).

It is, of course, a difficult matter to estimate just how much loss of power results from this abnormal condition; but if we consider the mechanism of the articulation and realize that the sole means of bringing the tibia forward, in the combined movement of extension with rotation, when the knee is flexed, is the quadriceps, and that the force must act through the patella, one can readily imagine that even a slight increase in the length of the anterior ligament must essentially disturb the normal action of the quadriceps upon the tibia as a whole. Add to this an abnormal or greatly increased lateral mobility at the knee, and a further disturbing factor is at once recognized.

The second condition—viz., a great degree of lateral mobility at the affected knee-joint—was also marked in Mr. J.'s case. The left knee had no perceptible lateral motion when the knee was extended and the quadriceps was relaxed. This lateral mobility, at the affected knee, was more marked from within outward, or in a direction which, if emphasized, would produce knock-knee. Measured on a large sheet of paper, by lead-pencil tracings, the femur being held, the heel moved through a very considerable arc, which, however, was difficult to measure in degrees. The execution of this lateral "wabble" gave rise to no pain, and, like the elongated anterior ligament, had not been detected by those who had previously examined the joint.

Comparing the conditions found in the affected joint with those on the other side, it did not seem difficult to explain Mr. J.'s frequent subluxations. The normal ligamentous relaxation, which occurs when the knee is flexed, is increased greatly in the condition above described. This alone would favor a slight tibial subluxation, and secondarily a subluxation of the semilunar cartilage, when, in an emergency, the quadriceps makes with the altered patella relation a sudden effort to apply the "air brake" in a semiflexed position. Perhaps the delay in clamping the tibia against the femur is measured, in time, by the merest fraction of a second, but it is enough to prevent a normal completion of the rotation corresponding with the stage of extension, and the cartilage is caught, pinched sometimes only, subluxated at other times, and in some instances "torn in ribbons," as related by Allingham.

The third condition in Mr. J.'s case was also present—*i.e.*, an inability to fully extend the knee. He was conscious, he said, all the time, when walking, that he "had a knee." Stretched out upon the floor the left popliteal space fell into its normal position, while the right was about one half inch from the floor. No voluntary effort could bring the limb straight, and force produced at first a most disagreeable sensation, and finally an acute pain. As Hey remarks, "The joint was like a gate with a stone caught in its hinges." There was also in Mr. J.'s case a marked atrophy of the muscles of the thigh, the calf being nearly normal.

From a study of this and my previous cases, it seemed to me that the indications from an orthopædic standpoint were (1) to correct the lateral mobility at the knee; (2) to prevent rotation of the tibia upon the femur; and (3) to so arrange the apparatus that extension of the knee would stop at a point where the traumatism of locomotion would cease to exist,—that is, at a point where pain and discomfort would be wholly eliminated. This meant an apparatus with simple antero-posterior motion, extending from the upper third of the thigh to the sole of the foot, and with a joint at the knee and ankle, the former having a mechanism by which extension would stop at the desired point.

I at once made a drawing of the apparatus which seemed indicated by the conditions above named. (See Figs. 1, 2 and 3.) In a few days it was applied. The relief afforded was instantaneous. All apprehension regarding the use of the joint was removed. Within a week the patient was walking four miles a day; within two weeks he was playing tennis, dancing, and riding his wheel, and he was in no way incommoded by his apparatus. It fitted so well that, under ordinary circumstances, it did not show through his clothing, and all pain and discomfort were removed. The apparatus was modified from time to time, especially to "coax" the joint into fuller extension, and the pad over the internal condyle was brought closer, thus further modifying the lateral mobility of the knee-joint.

At the end of six months he commenced to discard the use of the brace, at first in the house, afterwards for short walks out of doors, and finally, in the spring of 1892, he dispensed with the apparatus entirely. At my last examination there was normal extension of the knee, very slight lateral mobility, and the ligamentum patellæ had shortened nearly one quarter inch. The patient had neither discomfort nor disability of any kind.

It seems plain from this case and others which will be related that, in order to prevent the recurrence of a subluxation of the semilunar cartilage, it is necessary to correct the undue ligamentous weakness of the joint, and to prevent an abnormal rotation of the tibia; in short, to give the knee and ankle antero-posterior motion only. Under these circumstances undue strain is taken off the quadriceps and the ligamentum patellæ, and under favorable conditions the latter, as well as the relaxed crucial ligaments, may shorten very materially in the course of a few months.

Case II.—Miss P., aged twenty years, came under observation in May, 1892. Left knee: First accident occurred when she was eighteen, and resulted from a misstep in getting out of a carriage. It has occurred twice since, the last time while walking across an uneven surface in an uncultivated field. Had the usual treatment in each instance,—viz., a gypsum bandage, knee-cap, etc. Dreads repetition; is constantly on guard and always dreading any sudden movement; cannot fully extend knee; right ligamentum patellæ, two and a half inches; left, two; lateral mobility: right, marked; left, absent. It is three months since her last accident.

Apparatus applied in May, 1892; felt confidence and relief at once; was soon walking, dancing, and wheeling *ad libitum;* no inconvenience from apparatus, which with golf leggings does not show. The patient was so apprehensive of a relapse, and the brace was so comfortable, that she declined to remove it for experimental tests when I advised her to do so. She used it for two years, when, finally, it was discarded with complete recovery. No relapse. Lateral mobility about *nil;* full extension of knee, ligamentum patellæ about one eighth shorter than when treatment was commenced. Joint firm and serviceable in every way.

Case III.—Mr. L. N., aged twenty-eight years, in the fall of 1886, while playing football, sustained an injury to right knee; while giving a "rough tackle" to opponent, the entire weight of his antagonist came against the outer aspect of the knee in a slightly flexed position. "Felt a snap and went down like a shot." Ordinary remedies used; recovered in about three weeks. Six weeks later, while attempting to "kick a goal," the same accident occurred. In four weeks he had recovered. While wrestling in the early winter of 1888 same accident happened. Another month in bed with gypsum splint, etc. In April, 1888, while running to catch a "low fly" in baseball, he dislocated the cartilage for the fourth time in about two years. The usual time for recovery. In June, 1890, while simply crossing the right over the left leg, he produced the subluxation again. As simple as was the cause, recovery was the most tedious of the series, as he was on crutches for over seven months. Between 1890 and 1897 he was so far recovered that he was able to walk for quite a distance, to play golf, etc., without any recurrence of the accident. But he always felt that he must protect the articulation, and he instinctively kept it in partial flexion when walking. He avoided running, surf bathing, etc. On November 4, 1897, while getting off from a train with a dress-suit case in his right hand, the case was caught in the arm of one of the car-seats and thrown with considerable force against the slightly flexed knee, which was bearing his weight. The previous condition was reproduced. There was an almost immediate synovial effusion, with pain, heat, and limited motions of the joint. The resistance to full extension was marked, even after all acute symptoms had subsided, and he declined to rest at night other than on his back, so much did he fear that a turn in bed would bring on a fresh attack.

When I examined Mr. N., in November, 1897, just after the last accident, I found the following conditions: Right

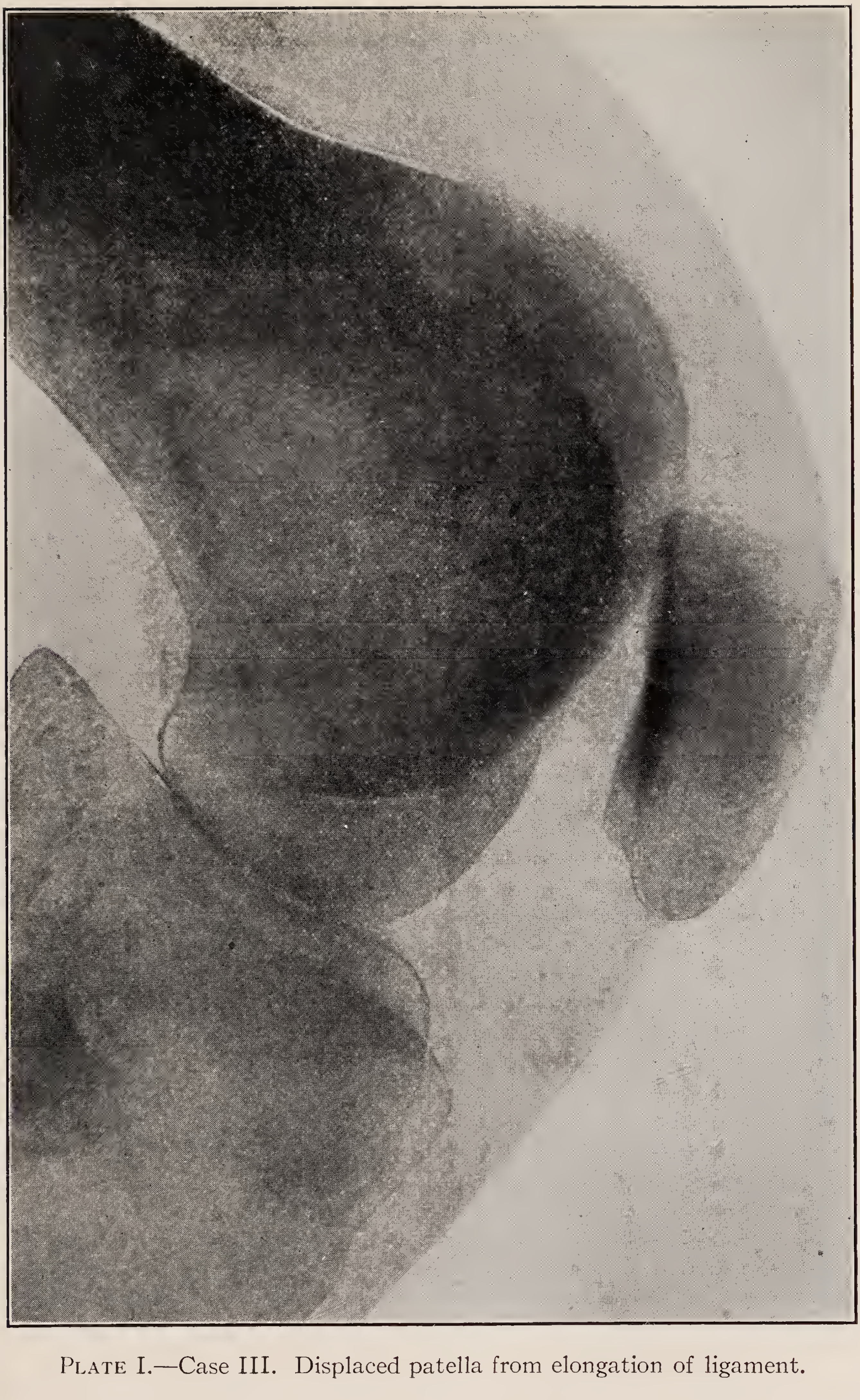

Plate I.—Case III. Displaced patella from elongation of ligament.

ligamentum patellæ, two and a half inches; left, two inches; two inches atrophy of right thigh; about one half inch atrophy of right calf. Lower extremities of equal length; very marked lateral mobility of affected knee; left knee rigid in full extension. Much pain, some heat, and marked synovial effusion.

While the acute effusion, etc., was subsiding, an antero-posterior splint for the leg was made. At the end of ten days the apparatus, limiting all but the antero-posterior movements of the knee and ankle, was applied. He at once walked with comfort and without a limp in this apparatus. In a few days more he was attending to his business, and at present he is able to walk, etc., at will. He has neither pain nor discomfort. He is still wearing his apparatus, and declines to consider any operative interference, although it has been suggested by me. (See Plate I.)

Case IV.—Mr. K. fell from back of wagon, which started up suddenly on November 11, 1893. Patient says, "My left foot reached the ground before I expected it to, and my weight came upon the bent knee sideways." Very severe symptoms followed; slow recovery; it was nearly two years before joint felt strong again. Had occasional "slips" and pains at various times, and on June 10, 1896, had effusion, with pain, after a decided "slip," as the result of over-exertion. On July 2, 1896, the patient had marked synovial effusion, considerable pain, walked with decided limp, and felt very much discouraged. Examination showed left ligamentum patellæ two and one quarter inches; right, one and three quarters inches; left knee great lateral mobility; right, none. Measured for apparatus and advised rest, ice, etc., during the construction of the brace. In about ten days apparatus was applied. The relief was immediate. The slight remaining effusion subsided under the protected use of the joint. Patient played tennis, danced, rode his wheel, played golf, etc., within a month from the time of using the apparatus. My last examination showed that though the ligamentum patellæ remained the same length, the lateral mobility was much improved, but was still present, enough so to warrant the further use of the apparatus, whenever the patient takes any unwonted out-door exercise. In the house he discards the brace. He has no pain or discomfort.

Case V.—Miss K. N., nineteen years old. In October, 1894, while walking across her own bedroom floor, on a level, hard-wood floor, patient slipped and nearly fell, and experienced a sudden "snap." "Something slipped in my left knee, and whatever it was slipped back again when I straightened my leg." Patient felt very faint; swelling and pain, with marked effusion followed. Joint quite stiff. Got well in a month, with rest, elastic knee-cap, etc. Was conscious of something wrong in the knee after recovery; could not fully extend the joint; was on guard all the time unconsciously. The following summer, while in bathing, standing sideways to an incoming surf, a comparative small wave struck the slightly flexed knee. She fell at once and was carried home. Rest, iodine, and a month in bed resulted in recovery. After second accident, going up and down stairs produced pain and often made the patient feel faint. In December, 1895, without any apparent cause, the subluxation was again produced. Water-glass splint this time, with prolonged rest; slow recovery. While romping with her sister in the summer of 1896, a slight lateral pressure on a semiflexed knee reproduced the subluxation. Rest, etc., resulted in a recovery in about a month. Patient consulted me in October, 1896. I found very marked lateral mobility of left knee; right normal. Left ligamentum patellæ, two and one quarter inches; left, one and five eighths inches. The tubercle of the left tibia was found to be outside of its normal position in this case, as if the tibia was rotated outward. Apparatus was at once applied, with a removal of all symptoms. Improvement was so rapid and was so great that it has been difficult to keep up a prolonged protection of the knee. The apparatus was worn spasmodically; very continuously for a week or two, and then discarded until a warning pain or a suggestion of a "slip" brings the patient to a realization of danger. Notwithstanding this, there has been a great improvement. The lateral mobility is much diminished; the knee extension is nearly normal, and patient dances, rides her wheel, etc., with her appara-

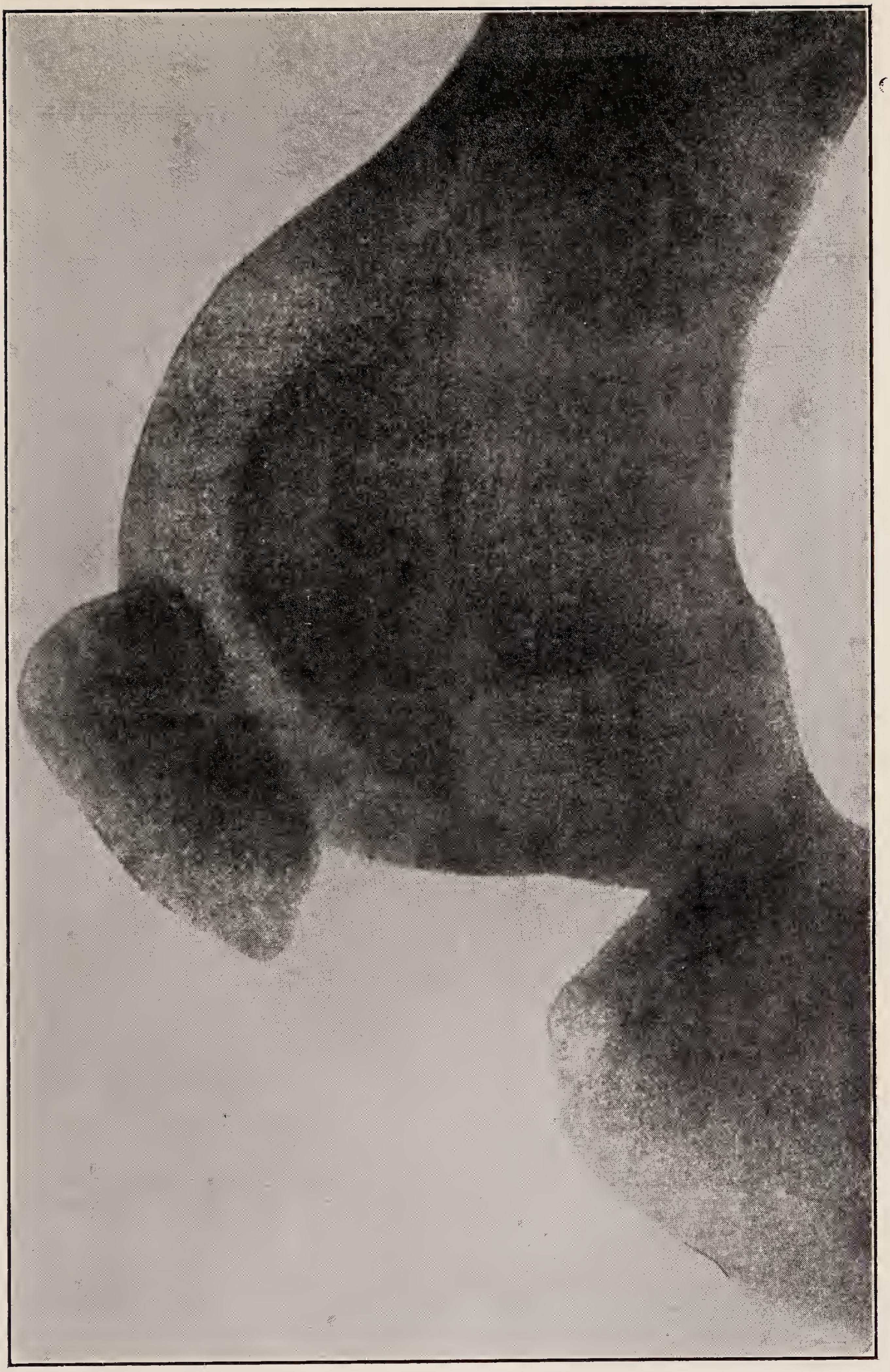

Plate II.—Case V.

tus on, without any discomfort or without fear of a relapse. (At the date of this writing patient has been without her apparatus for three months without any indication of a relapse, July 17, 1898.) (See Plate II.)

Case VI.—Mrs. K. Y. B. in November, 1895, was thrown down with right knee flexed and rotated out. Sustained a severe injury to right knee-joint, with a dislocation of the proximal end of the fibula. Had conventional treatment; got around in about a month; was advised to have operation to replace dislocated fibula. In the spring of 1896 made a slight misstep and produced a distinct dislocation of semilunar cartilage. The dislocated cartilage went back "with pistol-like noise" spontaneously. Patient felt a constant dread of a recurrence of the dislocation of the cartilage, she having had several minor "slips" which righted themselves.

I saw Mrs. B. first in February, 1897. A very painful joint. Right ligamentum patellæ, two and one quarter inches; left, two inches. Marked lateral mobility of injured knee. As in Case V, the tubercle of the tibia was outside of its usual position. Upper end of fibula very loosely held in its position, but not dislocated. Patient feels that it will become dislocated at every step, and seeks advice more on account of the tibial trouble than for relief of knee affection. After explaining the matter to Mrs. B., an apparatus was applied, limiting the knee and ankle to plain antero-posterior motion. Relief was almost immediate, and has been continuous. After six months patient commenced to go without apparatus. The knee symptoms have all disappeared and the tibia is securely anchored in its normal position; its ligaments having apparently shortened the same as do the ligaments of the knee under the protection from lateral or rotary strain. (See Plate III.)

Case VII.—Mrs. M. K. When twelve years old patient slipped in ordinary walking and subluxated the semilunar cartilage of left knee. This was followed by a prolonged hydrarthrosis and an equally long treatment, which resulted in apparent recovery. Had no further trouble (she is now twenty-nine) until last summer, when, in dancing, some one carelessly stepped on her dress. She felt something slip, lost all power over her knee, and fainted. Two months of rest, bandaging, massage, etc., resulted in recovery. In October, 1897, while playing golf, she made a slight misstep on uneven ground and felt the same "slip," which was followed by intense pain and utter inability to use the knee; was carried to her home.

When I saw the patient, in November, 1897, there was still marked effusion, an imperfect extension of the knee, considerable pain, a wide range of lateral mobility to the knee-joint, an elongated ligamentum patellæ (right, two and a half inches; left, two inches), and the altered relation of the tubercle of the tibia to the long axis of the femur, mentioned in the two preceding cases. Although the patient had pain in locomotion, she insisted on going about, and, after much hesitation, she decided to try the antero-posterior knee and ankle support. Its application was followed by an immediate relief, by a rapid decrease of the synovial effusion under rather extensive use of the joint, and by a removal of all fear of a recurrence of the "slipping," which the patient justly dreaded. She is now dancing with her apparatus on, playing golf, and using her knee, while at the same time it is on its way to complete recovery.

Case VIII.—Miss M. M., aged eighteen years, came under my observation in the spring of 1897. She gave the history of Hey's joint in both knees. The only case of the kind I have ever seen. The usual signs were present,—*i.e.*, the lateral mobility, the elongated ligament, the recurrent slipping, followed by pain, swelling, and disability. The right knee was less troublesome than the left, and there was a slight difference in the length of the patellar ligament, the left being slightly longer. On the left side the lateral mobility was very marked, being in excess of any other of my series. On the right side it was marked and about the same as in the cases above recorded. I applied the antero-posterior knee apparatus to either limb in this case, with the usual immediate relief. The braces were not worn continuously, the young lady being absent from home very much, but the relief has been

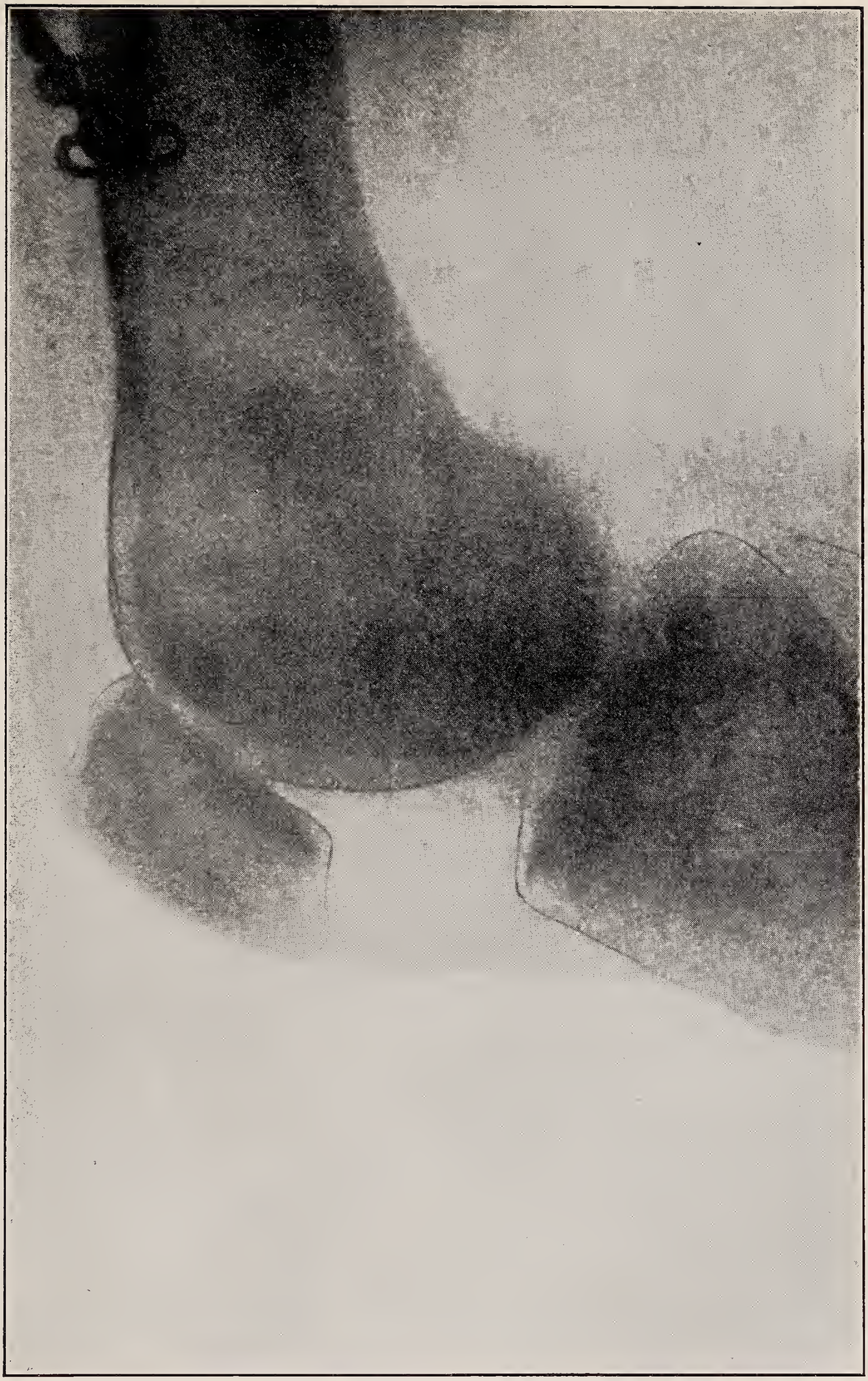

PLATE III.—Case VI. Displaced patella from elongation of ligament.

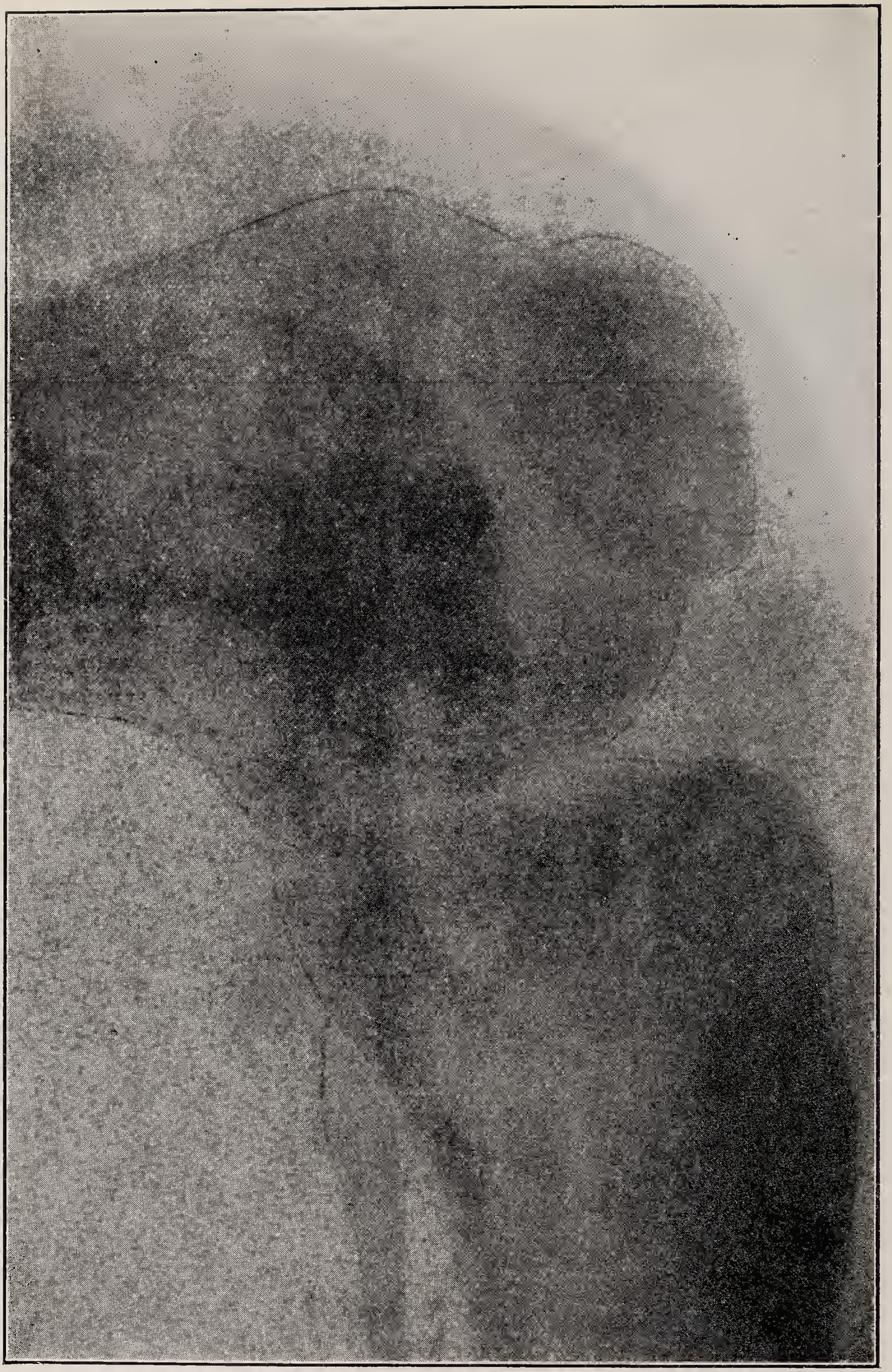

Plate IV.—Case VIII. Right knee.

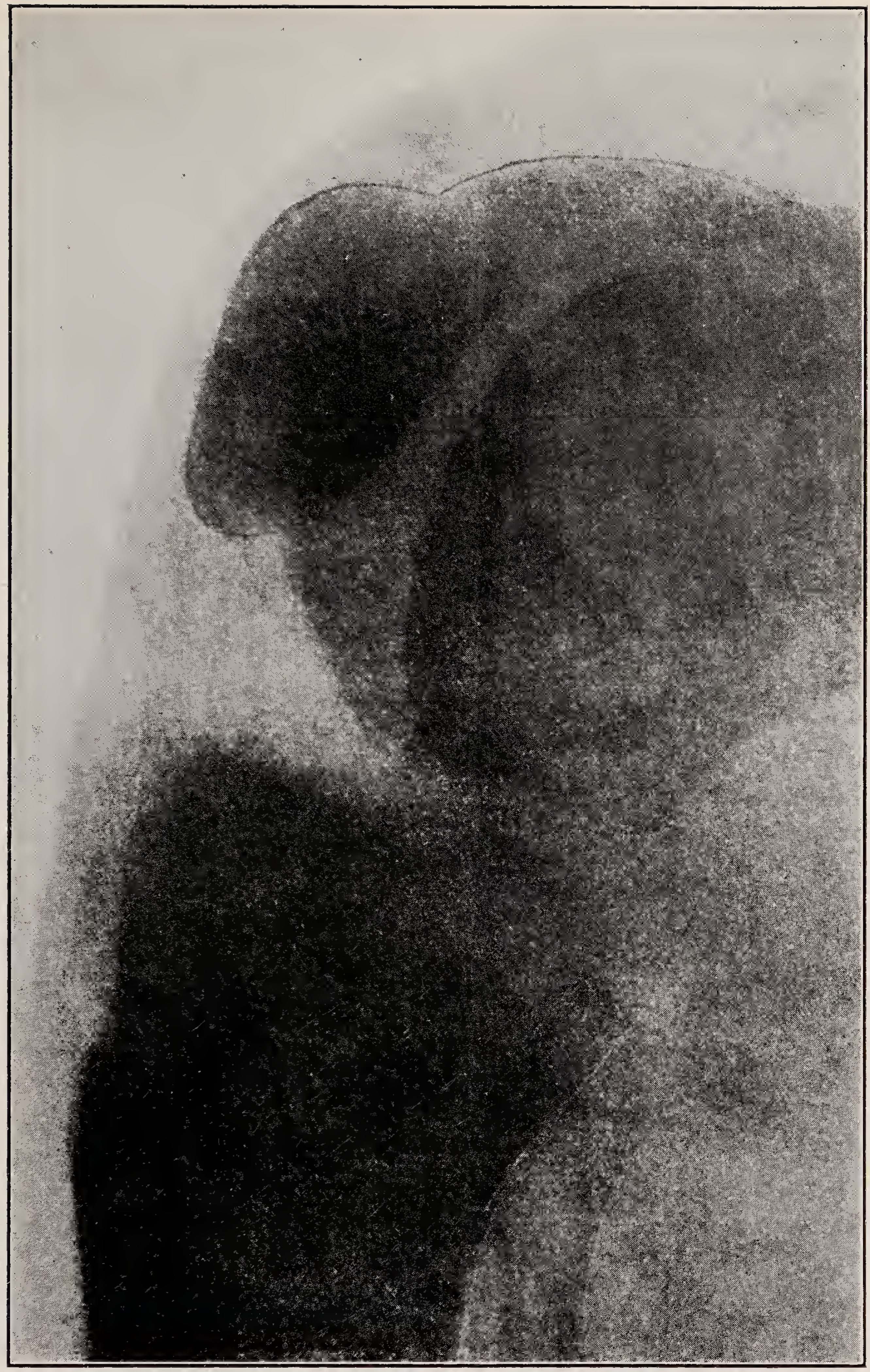

Plate V.—Case VIII. Left knee.

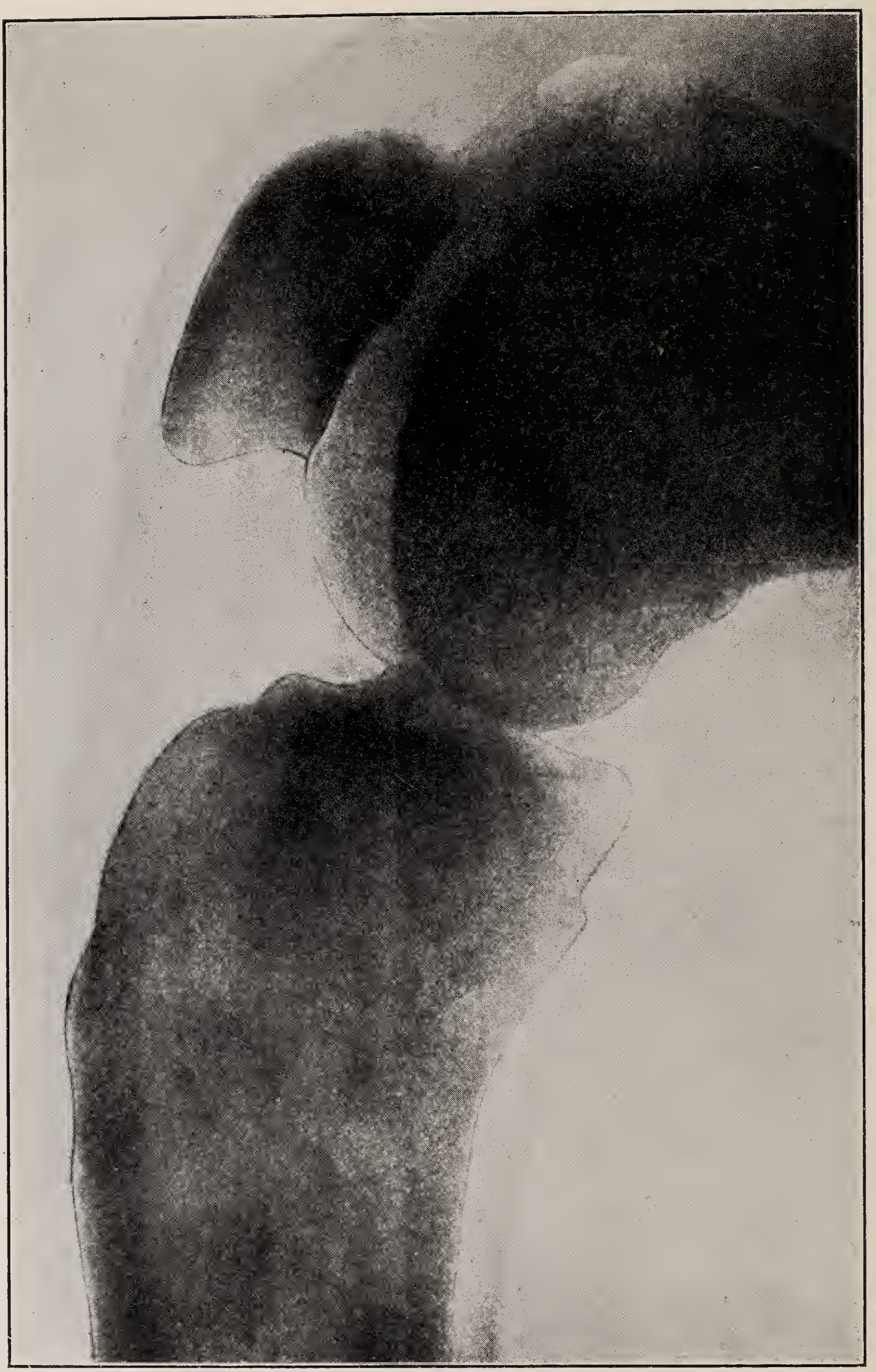

Plate VI.—Case IX. Left knee.

great. With the apparatus on the patient is able to go about at will, over rough ground, etc., and the improvement has been very marked. Latterly the apparatus has been discarded, and massage, etc., has been resorted to with no benefit. The braces have been again resorted to, and the question of operation discussed. It is likely that the operation, especially upon the weaker knee, would have been resorted to had not a very unfavorable report of the results of an operation in a similar case reached the ears of my patient at a critical moment. (See Plates IV and V.)

Case IX.—Miss G. M., sister of the foregoing patient, aged twenty years, sent for me last May with an urgent message that I come at once, as "in turning over in bed, in the early morning, something had snapped in the knee, which had become 'locked,' very painful and swelled." I went at once, and was able to feel distinctly the dislocated internal semilunar cartilage of the left knee. Flexing the knee, with some difficulty and pain, to about a right angle, and rotating the tibia outward, I gradually manipulated the dislocated cartilage into its normal position, with an immediate relief to all the painful symptoms. The knee-joint became immediately movable, except that it could not be fully extended. After a few days the swelling had all disappeared, and the patient resumed her ordinary exercises. The question of other than simple bandaging, etc., as a matter of treatment has been held in abeyance, pending an expected repetition of the dislocation, the patient electing this course in spite of other advice.

This is the only case in which I have actually seen and felt the dislocated cartilage, my other patients coming long after the original injury.

I might add that the brother of these young ladies has an occasional slipping of the internal semilunar cartilage, which, he says, has existed from boyhood. Thus far he has suffered only inconvenience from it. I have not examined his knee. (See Plate VI.)

Case X.—Mrs. B., aged twenty-six years, referred to me by Dr. Arthur A. Booth, of New York City, in April, 1898. Mrs. B.'s trouble dates back fifteen years. Her first attack kept her in the house for fifteen years. In 1892 she became ill, and was in bed from April until October, and during this period of rest the knee evidently became well, for upon recovery from her acute illness she had no further trouble until April, 1897, when the slipping occurred, and since that time she has had very much inconvenience, pain, and swelling. Until lately she was unable to walk over one block. The apparatus was applied and patient at once felt relief. She is now at her home in the country walking, etc., *ad libitum.*

Case XI.—Miss C. B., aged fifteen years, consulted me in April, 1898, with a history of "slipping patella." She had previously been under my care for ordinary flat-foot. A few days before I saw her, in April, without any apparent cause, the patella slipped, the dislocation being, according to the patient's description, inward, and being easily reduced by the patient herself. The accident occurred, sometimes, half a dozen times a day, but always when the knee was bent with the weight of the body upon it, except on some occasions when she was "climbing a tree or going over a fence." There was no special pain or swelling when I saw the patient, though the joint-outlines were altered and the fossæ on either side of the patella were modified. The ligamentum patellæ was longer than its mate by a full quarter of an inch, and there was present an unusual degree of lateral mobility, such as seen in the cases reported above. An apparatus, such as was used in these cases, was applied, with immediate relief. At first the patella would slip occasionally, but recently, since the support was more accurately adjusted and the pad on the inside of the knee was closely adjusted to the internal condyle, the dislocations have entirely ceased, and the lateral mobility has become modified. I expect this patient to recover in a few months without any operative interference.

I call attention, in this case, to the lack of union at the epiphyseal line at its anterior aspect, as shown in the skiagraph. This may or may not be a contributive factor to the slipping patella. (See Plate VII.)

In order to compare the patellar conditions in a normal knee-joint with those shown in the accompanying skia-

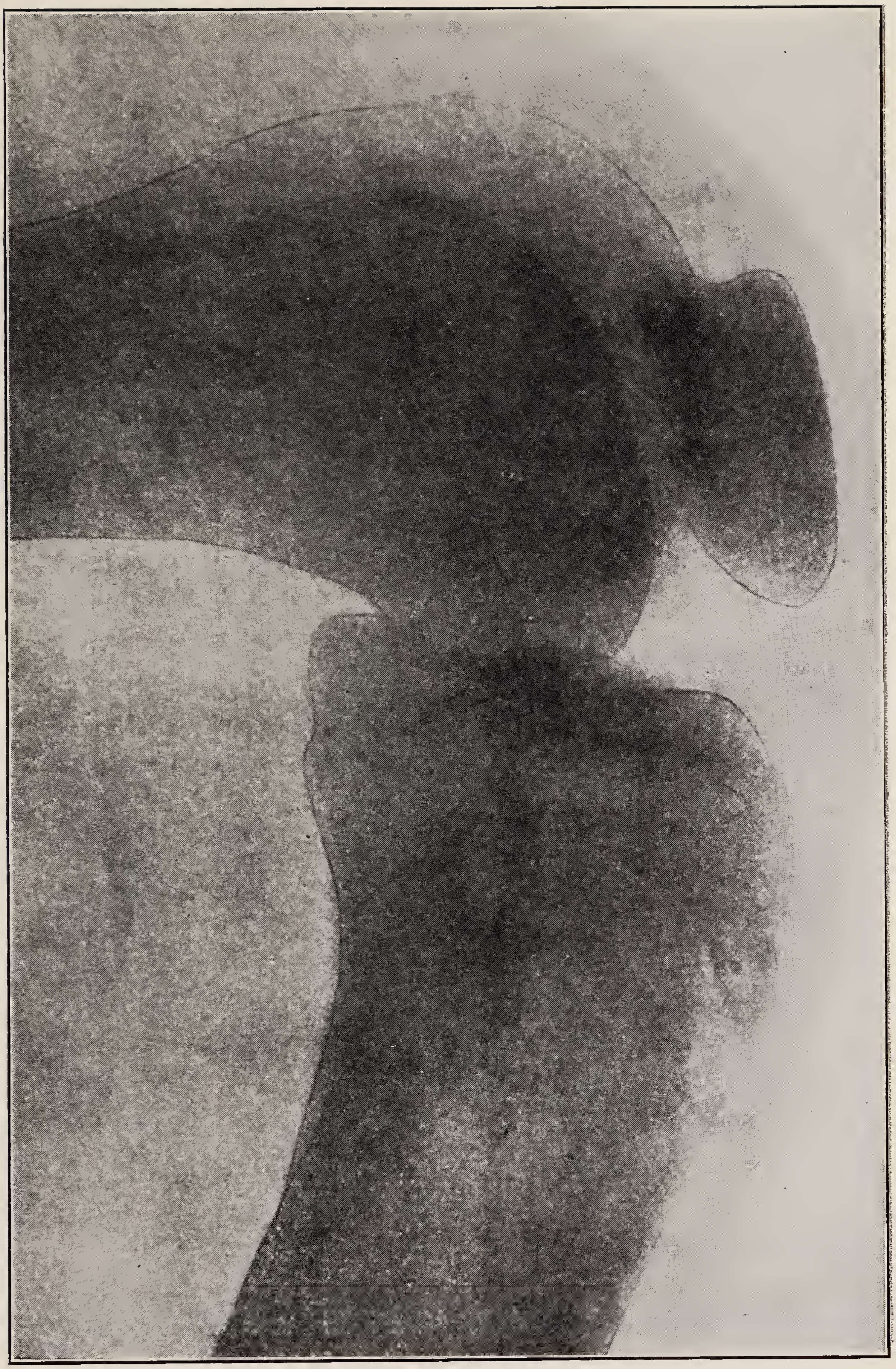

Plate VII.—Case XI. Slipping patella in a girl of fifteen years; delayed epiphyseal union.

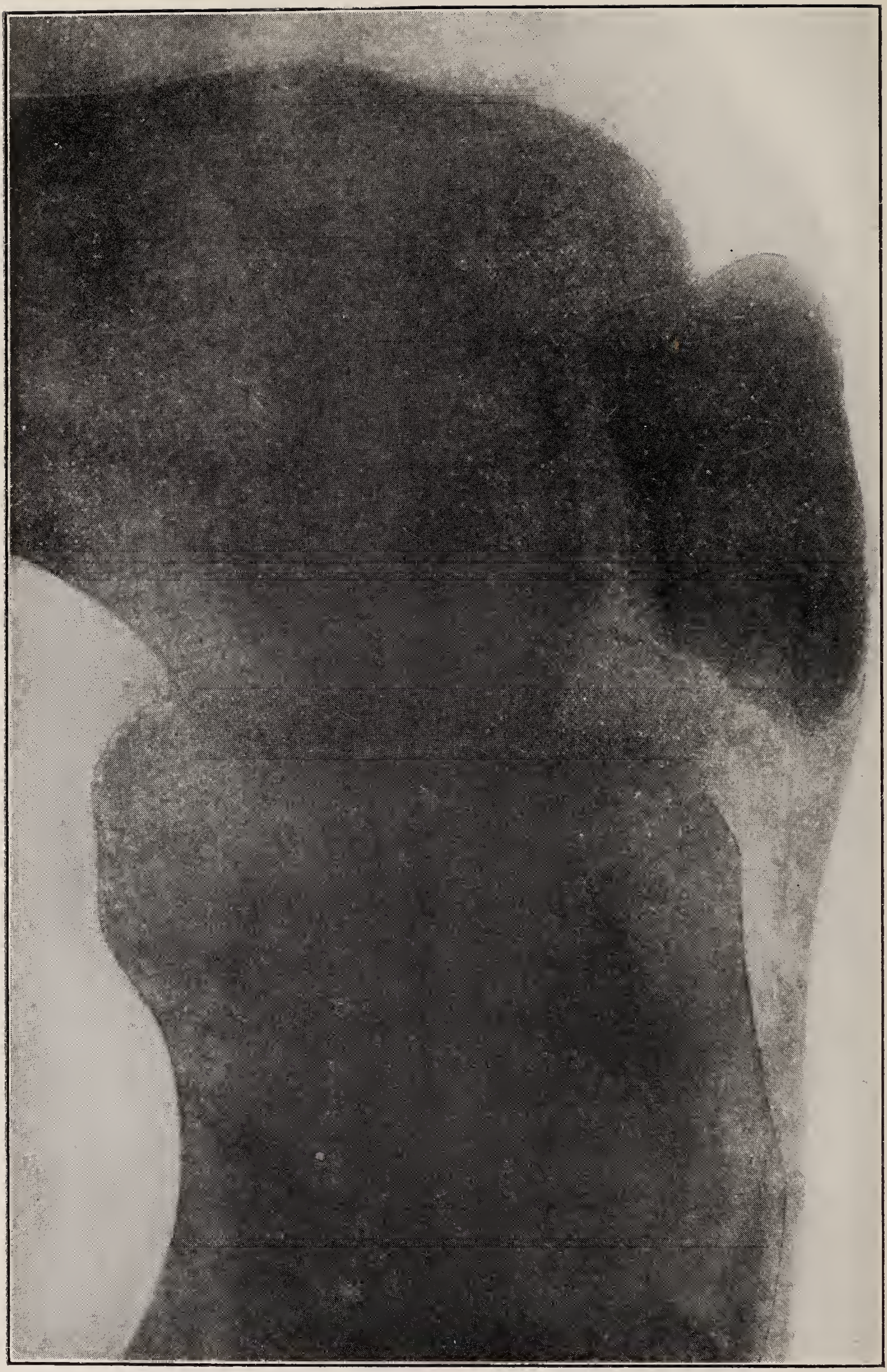

Plate VIII.—Normal patella of a healthy, active adult male, aged twenty-six years.

graphs of Hey's joint, I have had an X-ray picture made of a young man, aged twenty-six, with perfectly normal knee-joint. This will be found in Plate VIII.

The location of the patella in these radiographs of the normal knee is clearly shown. It will be observed that the patella lies well down in the trochlea and in close proximity to the head of the tibia. On the other hand, in all the cases of dislocated semilunar cartilage here shown the patellæ are drawn upward, and in some cases the lower end of the knee-cap is tilted upward away from the trochlea. In every case the elongation of the ligamentum patellæ is apparent, and the modified action of the quadriceps muscle can be readily appreciated.

In Figs. 1, 2, and 3 the apparatus used is shown. It is a modification of an apparatus I first applied to a young girl over ten years ago with knee-joint disease, whose name is Campbell, and I named the apparatus the "Campbell traction knee- and hip-splint," after the patient to whom it was first applied. In the present instance the traction rod and pelvic band are removed, the object being not to produce traction, but to prevent every movement at the knee and ankle except antero-posterior motion. In short, to turn the knee into true hinge-joint, removing entirely the rotation of the tibia. In Fig. 1 the apparatus appears unattached to the leg and thigh. The important part of the apparatus, next to its simple hinge movement, is the joint at the knee, which is so arranged that it will stop the extension just at the point of comfort to the patient, and this point of comfort represents an absence of strain upon the knee-joint ligaments. This is very essential to the cure of the trouble, for experience proves that if the strain is taken from the ligaments they will shorten, and the "wabbly" knee will gain stability and strength in a few months.

Figs. 2 and 3 show the apparatus applied, and needs no comment or description. It is important that the center of the pad at the knee should be opposite the true center of motion (opposite the most prominent point on the internal condyle is near enough) at the knee, and that it should rest snugly against the condyle without undue pressure. The apparatus need not be made heavy, the principal strength being necessary in the rod which connects the knee with the ankle-piece. It is preferable to have this rod on the outside.

I might mention several other cases and facts bearing upon the points raised by the foregoing histories. Those mentioned, however, are sufficient, I think, to establish some facts in relation to the questions raised; and these are, briefly:

(1) In many cases of Hey's joint there is an acquired or, perhaps, congenital lateral mobility of the knee-joint. This condition existing, the normal rotation of the tibia in flexion or extension of the knee is greatly increased.

(2) In many cases, if not in all cases, there exists an elongated ligamentum patellæ, which so modifies the action of the quadriceps extensor muscle upon the tibia that the force of its contraction upon the tibia is modified or delayed in such a way that extension and rotation are not synchronously performed. And it seems more than probable that this condition forms an important factor in the production of the subluxation of the semilunar cartilage.

A simple elongation of the patellar ligament may exist, and frequently does exist, with no outward symptoms except those mentioned in my article "On Elongation of the Ligamentum Patellæ as a Factor in the Production of Certain Knee-Joint Troubles and Difficulties in Locomotion" (*New York Medical Record*, January 16, 1892). I may quote the following brief extract from the paper as bearing on this point:

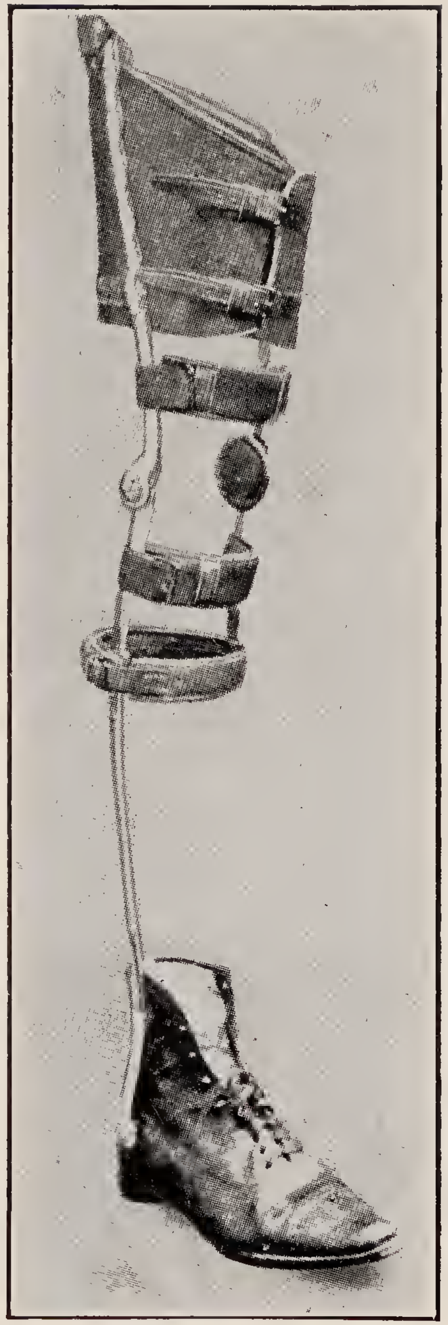

FIG. 1.—Apparatus for Hey's joint; antero-posterior motion only at knee and ankle.

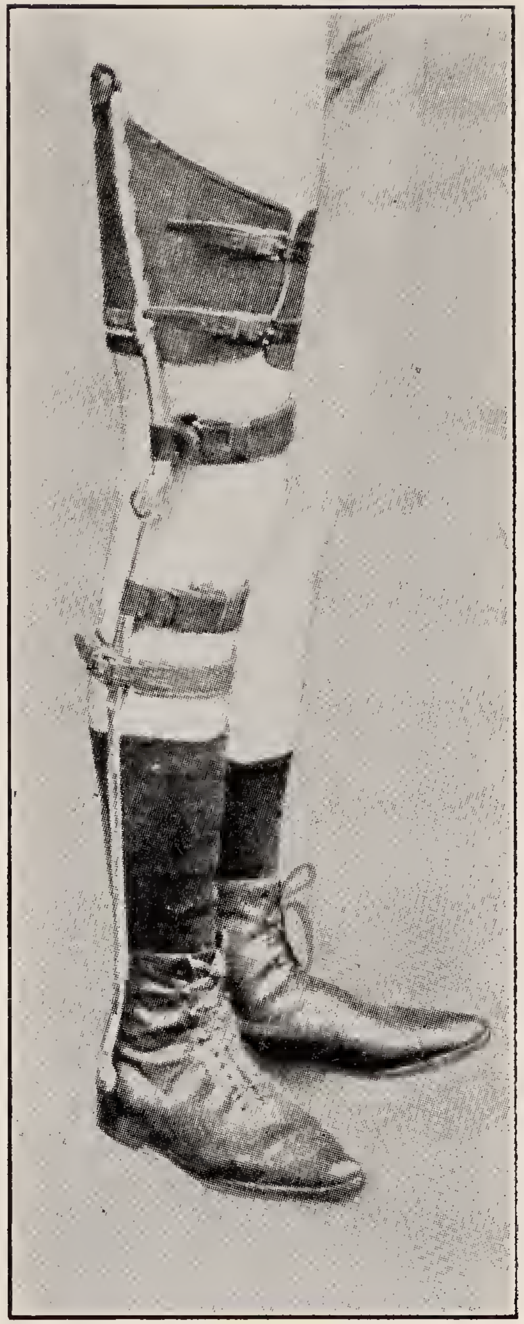

FIG. 2.—Apparatus for Hey's joint applied; lateral view showing "stop joint" at knee.

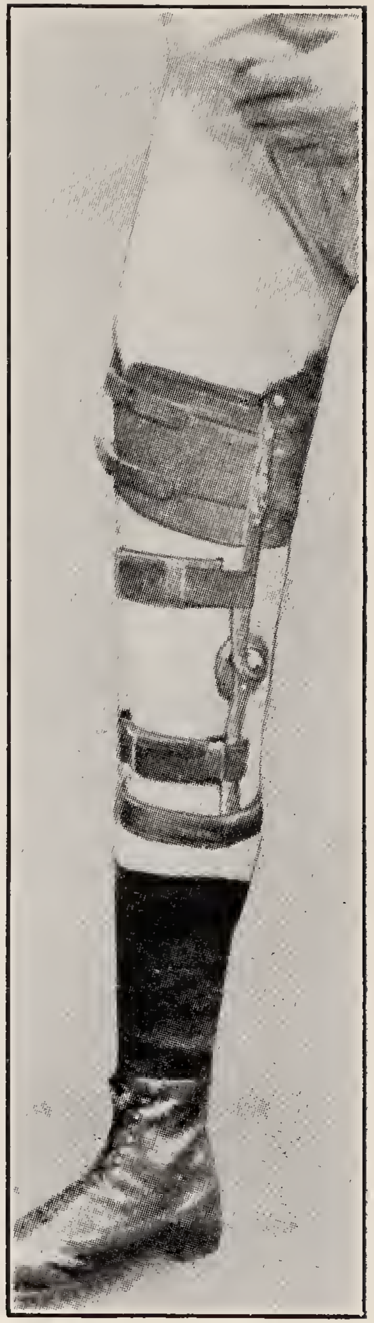

FIG. 3.—Apparatus for Hey's joint applied, showing protective pad inside of knee.

"I might relate other interesting cases where there was knee trouble due to the lengthened anterior patellar ligament. In some of them there was heat, pain, and swelling, due to overstrain, but no true joint inflammation. In others a sense of weakness and debility. In others a tendency to dislocation of the patella outward. In all, however, there was a greater or lesser degree of disability under severe, or in some cases ordinary, use of the joint. It was especially noted where much strain was brought upon the articulation in the flexed position."

The various operations for "slipping patella" which have been devised are more or less based on the conditions described in my article, which, so far as I know, is the first stated contribution to this subject.

While, therefore, elongation of the patellar ligament may exist and result only in disability, discomfort, or a slipping patella, it would seem that a further structural weakness of the knee was necessary to produce Hey's joint; and in all my cases, without exception, there has existed, to a marked degree, either a traumatic or an early acquired, lateral mobility of the joint, due perhaps to an infantile rachitis, or to some other cause disturbing bone and ligamentous nutrition in infancy, or in pre-adolescent life.

On the other hand, a considerable degree of abnormal lateral mobility may exist at the knee, with a normal ligamentum patellæ, without the danger of subluxation of the semilunar cartilage. It would seem as if both of these conditions were necessary to produce the trouble, except, of course, those cases like L. N., where the first trouble was produced by the application of an extreme degree of force. And even in this case the abnormally elongated ligament was a probable factor in the production of the condition.

I cannot close this paper without quoting from Hey's original article, which is a model of clearness and conciseness. He says ("On Internal Derangement of the Knee-Joint," Hey's *Surgery*, printed by James Humphreys, Philadelphia, 1805), "The disorder may happen either with or without contusion. In the latter case it is readily distinguished. In the former the symptoms are equivocal till the effects of the contusion are removed. When no contusion has happened, or the effects of it are removed, the joint, with respect to its shape, appears to be uninjured. If there is any difference from its usual appearances, it is that the ligament of the patella appears rather more relaxed than in the sound limb. . . . The patient himself cannot freely bend nor perfectly extend the limb in walking, but is compelled to walk with an invariable and small degree of flexion."

This most acute observer, under the term "relaxation of the ligament of the patella," calls attention to a condition which I have since called "elongation of the ligamentum patellæ."

Even with the clue given by Hey, no one that I know of has studied this feature of the condition, and as I had not seen Hey's original article until after I had commenced to write this paper, it may be taken as an independent observation confirmatory of the statement first made by this eminent observer.

What Hey saw and thus accurately described, we can now demonstrate with the Röntgen ray.

In Allingham's treatise, already referred to, he says: "A lax condition of the ligaments about the joint, caused by general debility or previous synovitis, predisposes to these accidents, on account of the insecure way in which the tibia and femur are bound together." Again: "The injury is caused by some sudden and almost involuntary movement. Most of the muscles governing the joint are then thrown off their guard or fail to act in concert one with the other."

But I do not agree with Mr. Allingham when he says, "All this may occur even in a healthy joint," if for healthy we substitute "normal," except under circumstances where great lateral pressure is applied, and when a true subluxation of the tibia occurs as a result of direct violence. In several of my cases there was no direct violence. In these cases there existed a condition which I have attempted to describe, and which directly favored the subluxation. And further, the muscles are not "off their guard." The quadriceps acts promptly, but the patella instead of clamping the femur with a normal length of ligament in the trochlea is tilted upward at the lower end, and the muscular effort is so delayed that the movable semilunar cartilage is caught, perhaps only slightly pinched, or really dislocated, and sometimes seriously damaged.

And my experience proves conclusively that Mr. Allingham is wrong when he says, "The apparatus . . . is cumbersome; but feebly efficient; it cannot cure the affection."

I would rather say of Hey's joint, as well as of many other conditions, that the surgeon would not operate so frequently if mechanico-therapy was as well taught and practised in our medical colleges as is the operative surgery of the present day.

:o:

NOTE BY THE AUTHOR

(September, 1923)

All the patients who remained under my care and followed my instructions were permanently cured, including two who had previously and unsuccessfully submitted to an excision of the cartilage. It is an easy task to remove the offending cartilage—but the operation does nothing to restore the stability of the joint.

ON THE TREATMENT OF ACUTE AND SUB-ACUTE INFLAMMATION OF THE KNEE-JOINT BY APPARATUS PERMITTING LOCOMOTION WITH PROTECTED ANTERO-POSTERIOR MOTION AT THE JOINT[1]

Presented at the eighteenth annual meeting of the Association at Atlantic City, N. J., June 8–10, 1904.

THE speedy relief and comfort which follows the application and use of the apparatus described by the writer in the *Annals of Surgery* for October, 1898,[2] in cases of subluxation of the semi-lunar cartilage at the knee-joint, are familiar to all who have pursued this plan of treatment. It is not unusual for a patient with a subluxated cartilage to resume at once, and without pain or fear of a recurrent displacement, all his usual habits of life and activity. A recent case coming under my observation and treatment will illustrate how speedy and complete this relief may be:—

A boy of seventeen, an inmate of one of our popular boarding-schools, while playing football last autumn, received a severe injury to the right knee. With the knee in a slightly flexed position, it sustained a severe impact, the force being lateral and from the distal side. Immediate disability followed, with heat, pain, and swelling. When

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[1] *Transactions of the American Orthopædic Association*, 1904. Vol. xvii, p. 98.

[2] "On the Cause and Mechancial Treatment of Subluxation of the Semilunar Cartilage of the Knee-Joint," *Annals of Surgery*, October, 1898. *Vide* preceding essay.

I saw him about ten days later, there was still a marked effusion within the capsule of the joint, a distinct protrusion at the joint line between the inner condyle and the head of the tibia, and a very marked lateral mobility. The patient was unable to walk without crutches. Indeed, he was unable to bear his weight upon the injured member. Within four days I had applied the apparatus described in the *Annals of Surgery* with the joint about to be described, and within a week he was walking without any support except that afforded by the apparatus. Within two weeks he was dancing and riding the bicycle. After eight months' treatment his recovery has been uneventful and complete.

With a variation which is individual rather than pathological, this is the history of many patients treated during the past few years. The ages of the patients vary from fifteen to fifty-nine years. Both sexes—with the female sex in the majority—are represented; and it includes as well a considerable number of women who may be described as portly, one patient weighing about one hundred and ninety pounds.

It is not my intention to review at length the subject of the mechanical treatment of semi-lunar dislocations, as I have already done so on a previous occasion.[1] Almost without exception those patients recover who follow the surgeon's advice, who afford him co-operation, and who give him an opportunity to frequently revise and modify the support. Without this, failure is apt to occur; for an inefficient support is probably worse than none at all.

The following incident, occurring to a patient under treatment for subluxated semi-lunar cartilage, makes it apparent that the apparatus described in the *Annals of*

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[1] *Op. cit.*

Surgery has a much wider range of applicability than that for which it was originally intended:—

Mr. A. S., aged eighteen, who had been under observation and treatment for about three months for subluxation of the semi-lunar (internal) cartilage, had occasion to attend a dance one evening, in connection with his graduation from a popular private school, prior to his entrance to college in 1901. He removed his apparatus to attend the dance, contrary to my instructions. During an interval between the dances, while "sky-larking" with some friends upon a winding staircase, he slipped and fell, producing an immediate recurrence of the dislocation of the cartilage. He got home with some difficulty, and came to me the next morning on crutches, bringing his apparatus with him.

The knee-joint was very much distended, with an effusion. It was hot, painful, and tender. I advised the boy to go home, where I promised to soon follow him and apply a plaster-of-Paris splint. He stoutly maintained that this was impossible, as he was obliged to attend his college entrance examination that day, and that he had brought his brace with him, hoping that I could adjust it in some way to meet the exigency of the occasion. There was no time to apply a gypsum splint. He was on his way to the examination hall even then. The condition of affairs seemed urgent enough to warrant my complying with his request. With the aid of wrenches the outline of the brace was made to conform with the greatly altered joint, and the apparatus was applied. With the support afforded by the crutches he attended the examinations for three days.

At the end of that time the patient had experienced much relief, although he had bent his knee for several consecutive hours at the examinations, and had used it much as he otherwise would have done, had it not been affected, except that he used his crutches in walking. The effusion, pain, and heat had subsided very considerably. He walked across the floor of my office with only a slight limp, in his apparatus, without his crutches. The "pin" inserted in the joint to arrest motion just inside of the point indicated by Nature was, however, too short to be fully effective. While he waited in my office, this pin was made longer. With this change he experienced no pain, and walked better. Indeed, he experienced so much comfort and relief that, as a matter of test, I directed him to go without his crutches, to walk moderatly, and report to me the next day.

Rapid improvement followed. He soon was walking about the streets, with a gradually decreasing effusion; and at the end of about two weeks, and after he had commenced walking as above, the effusion had disappeared. He went into training for a running match soon after he was discharged as cured the following spring, and he came out second in the race.

Mr. H. R., aged twenty-two, while attending a football contest, as a spectator, at New Haven in October, 1901, experienced—without any apparent cause, except a slight slip—a violent pain in his left knee, and was with difficulty conveyed to his home in New York. I saw him the evening of the accident, if such it may be called. The patient had, about ten years previously, been under my care for hip-joint disease upon the opposite side (right), from which he had recovered with slight deformity and limited joint motion.

The history developed at the time of my examination showed that for several years he had noticed that the left knee was occasionally quite "puffy" and painful, and that there were times when he was obliged to favor it. He had not, however, consulted any surgeon regarding his condition, and had allowed the matter to go on, hoping for a spontaneous cure. The knee had been troubling him for several days prior to his New Haven trip, and he had attended the game largely on account of guests whom he had invited. He described the attack as coming on very suddenly, and with great pain, and without any apparent cause, except, as above stated, a slight misstep. Certainly there was much disturbance in the joint. The capsule was so distended as to suggest some dense, semi-solid substance; and I could not satisfy myself that the contents of the capsule were fluid until the use of the hypodermic needle demonstrated the existence of a clear serous fluid. I applied the usual remedies locally, the heat being combated by ice. The patient remained recumbent. No splints were employed. The effusion and heat diminished very gradually. About three weeks after the first symptoms I applied the apparatus described in the *Annals of Surgery*, there still being considerable effusion and some heat, and about one-half of normal flexion in the joint. The "pin" was carefully located to arrest motion just where all strain would be taken from the crucial ligaments, and the patient was instructed to walk. Inside of a week he was walking about as much as was customary with him; and on December 19 he stopped treatment, being, as he said, so well that he did not need any surgeon. He wore the brace for another month or so, when he discarded it. The effusion has not returned; and the joint, about a year ago, was normal to all ordinary tests.[1]

I could add to these histories many others of patients who have recently been under my care. They all give the same history as regards the immediate and also the permanent relief—even in cases of acute synovitis—afforded by the use of the "pin-stop" apparatus, placed as it is in the eccentric joint of the brace. As soon as the really acute symptoms become modified, and long before the effusion disappears, the support can be applied.

The use of this protection to the knee—the introduction of this "stop" which saves the ligaments from strain, and which supplies to the unprotected knee an artificial glenoid cavity, or an olecranon process—serves again to re-enforce my statement that in joint inflammation "within the limit set by Nature, motion, I believe, to be harmless."[1]

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[1] "*June 23, 1904.*—An examination of the patient to-day shows very marked improvement. The swelling and thickening about the knee are very much modified. The patient can now quite fully extend the knee-joint without pain, and the arc of flexion has increased over 20°. The patient walks about, attends to business, and is most comfortable in the apparatus, which causes him much comfort and no annoyance. The prognosis is most favorable."

In chronic synovial disease of the knee I have applied this apparatus only once. In this case, a young girl of fifteen, with a history covering ten years, and with about 40° of motion, the application of a "pin stop" has been followed by relief from pain, a cessation of limping, a modification of the morning stiffness, and an increase of the antero-posterior motion at the joint. The swelling has also become reduced,—about one-half an inch in six months.

In an adult with a history of several recurrent injuries, covering several years,—with a strong suggestion of a joint inflammation of a chronic nature, but not apparently tuberculous,—relief has not been marked. The joint motion, however, has increased under the use of the "pin stop," and the apparatus gives much comfort.

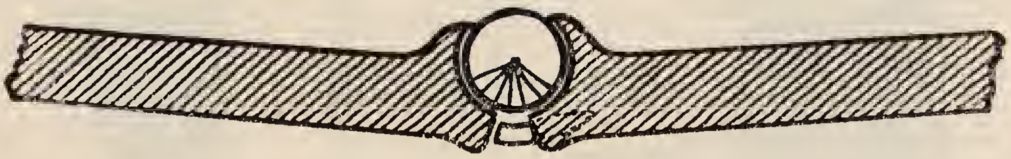

FIG. 1

The apparatus has been fully described in the *Annals of Surgery*.[2] It is not intended to produce traction, as such, "but to prevent every motion at the knee and ankle, except antero-posterior motion. In short, to turn the knee into a true hinge joint, removing entirely the rotation of the tibia. . . . The important part of the apparatus . . . is the joint at the knee, which is so arranged that it will stop the extension just at the point of

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[1] *Transactions American Orthopædic Association*, vol. ii, p. 100.

[2] See also p. 493 of this volume.

A LECTURE ON KNOCK-KNEE AND BOWLEGS[1]

THE conditions which I propose to discuss are interesting in many ways, and as the principles involved in their mechanical treatment are so nearly identical, I have thought it best to include them in one article.

The condition known as knock-knee or genu valgum is one that is well known to all. Like club-foot and other strictly-speaking painless deformities it is easily diagnosticated, and I need not stop to describe the signs by which we recognize a well-marked case of this malposition. It, as well as bowlegs or genu varum, has been, until quite recently, very much neglected by surgeons, for it is not many years ago that this class of cases were almost invariably referred by the medical profession to the instrument maker, who seemed to rest content with placing the deformed members in the most cumbersome and not infrequently the most expensive apparatus attainable.

But the recent progress made in the study of the causes and pathology of chronic deformities, and the great interest shown by general as well as by special surgeons in the scientific means of relieving them, and more than all the evident disposition on the part of the medical profession to realize that such conditions as they refuse to reach scientifically become sooner or later the legitimate prey of charlatans, have brought about a much different state of affairs. These statements are particularly sustained by the attention which the conditions under consideration are now receiving from surgeons of both hemispheres, and the abundant literature which exists upon the subject of their treatment. It would seem as though the matter of operative treatment was at present very much overdone, while the questions of cause and ultimate results have not received the attention they demand.

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[1] *American Journal of Obstetrics and Diseases of Women and Children*, July, 1881.

The temptation to accomplish the brilliant in chronic surgery is very enticing. We certainly are not going too far when we remark that no surgical operation can be accepted by the medical profession, however brilliant the *immediate* results may be, until sufficient time has elapsed to test its *ultimate* results. See for instance the incorrect impressions produced not many years since by the excellent results said to follow excision of the hip-joint. This is only one illustration of the bad practice of prematurely publishing results, the greater part of which are imaginary, or, at best, only temporary. In orthopædic surgery especially, which represents surgery of the most chronic type, we frequently study symptoms from month to month, or even from year to year, while in acute surgery we watch from hour to hour or from day to day. What seems to be a good result in chronic surgery to-day may be a very bad one in six months or a year, or even in ten years. We must not ignore these facts in considering the results obtained by methods of treatment which are as yet only experimental as applied to genu valgum and genu varum. And this should be a guide in considering methods of treatment which have not stood the test of years in orthopædic surgery. Never expect to accomplish in chronic surgery the rapid effects which are obtained in acute surgery. In acute surgery the result, recovery or death, occurs soon. In chronic surgery, and in orthopædic surcomfort to the patient; and this point of comfort represents an absence of strain upon the knee-joint ligaments."

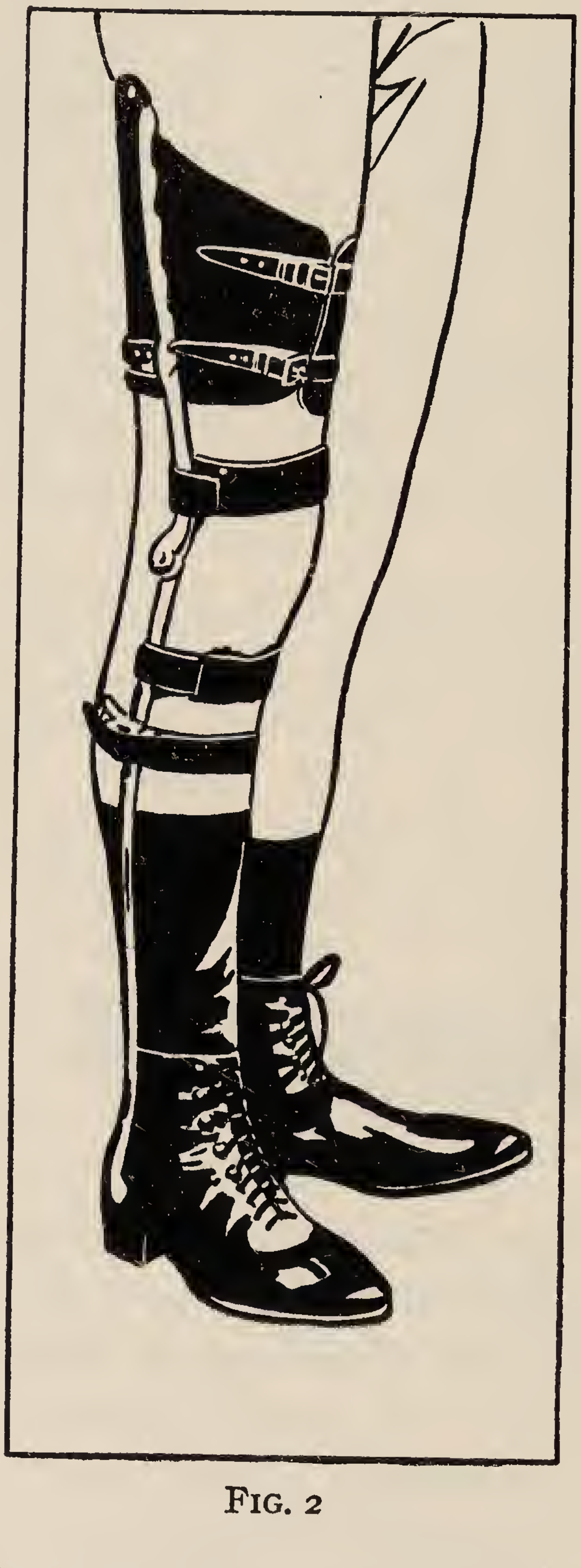

FIG. 2

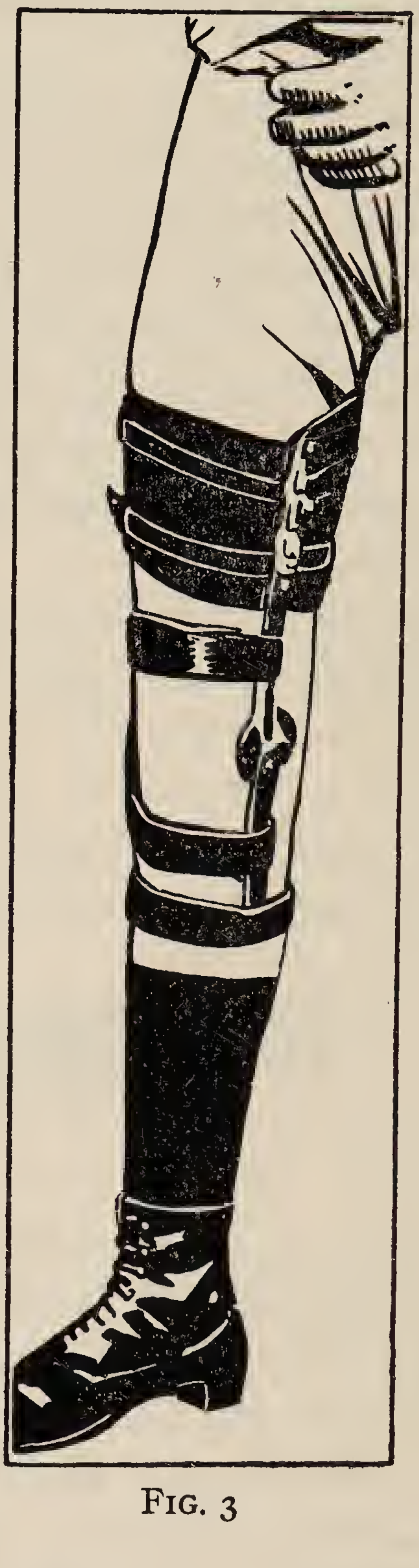

FIG. 3

In order to demonstrate the "pin stop" part of the apparatus, which is the adjustable "olecranon" of the knee-joint, I submit herewith a drawing showing it. (See Fig. 1.) It is placed in the outside bar of the brace in the upper part of the eccentric knee-joint of the apparatus. The center of motion in the apparatus is placed half an inch, more or less, back of the long axis of the outside bar. Into the "jaws" of the joint thus created (see Fig. 2) is inserted a pin curved to meet the arc of the artificial center in the knee of the apparatus. The "pin stop" should be made long, so that the surgeon, in adjusting the apparatus, may gradually file it down until the point of contact of the pin with the lower half of the joint is the one required. This point of contact should not only represent comfort to the patient, it should stop at a point where there is an audible "click" with each step the patient takes. The apparatus as applied to a patient is shown in Figs. 2 and 3. When this is accomplished, we may know that our patient is safe, and that he can walk, even with an effusion within the capsule, with a better prospect of cure than with the ever-ready and convenient but, as I believe, the very inefficient plaster-of-Paris splint.

NOTE BY THE AUTHOR

(September, 1923)

It is the "pin stop" which makes the apparatus so efficient. As is well known one can stand erect without muscular effort at the knee. The "pin stop" arrests extension of the joint at a point which prevents actual contact between the femur and tibia. It removes inter articular pressure, and strain on the ligaments is much modified.

gery especially, I do not hesitate to say that more deaths are produced by meddlesome interference with the knife or otherwise than by the disease itself. Conservatism in orthopædic surgery, experience teaches me, is absolutely necessary. Operative surgery in orthopædic practice is, or should be, almost wholly unnecessary. Even tenotomy may be avoided in the treatment of club-foot, and in some conditions where contracted muscles exist, such, for example, as are found in chronic joint disease, we find that ether removes the contractions. The conditions which require the knife in orthopædic surgery are rare. They are, as a class, confirmed deformities which have been either maltreated or neglected. If students were taught in our colleges how to recognize the earliest stage of progressive deformities of all types, this necessity would be further removed. And I have made it my rule, and it is a good one, never to use a knife to remove deformity when I can avoid it. We have no right to say: I can accomplish the *removal of the deformity* quicker by its use. The question we must ask ourselves and answer candidly is: Will the ultimate usefulness of the member or members be greater if I remove the deformity quickly and merely save time thereby? We have no right as conscientious surgeons to think of time or effort in orthopædic practice. We should be willing to spend all the time necessary and to use all the effort required to obtain the best ultimate result. Fortunately there are quite a number of operative procedures in orthopædic practice which do not imperil life, and in these cases I doubt not there will always be some difference of opinion as to whether it is best to interfere and hasten the reduction of the deformity by cutting. But there are other conditions where what may be called capital operations are performed in cases where the result, I am very sure, would be better if nature were simply assisted. Here there can be no doubt, it seems to me. My own experience leads to only one conclusion in this connection, and that is, that we can secure in all but the very exceptional cases much better results by imitating nature and using gentle rather than forcible means. In the painless but progressive deformities of the limbs which are accessible to operative surgery, such as the conditions now under consideration, the cases are rare, in my experience, that need the knife; still, as from neglect or bad treatment the condition may become so confirmed as to require operative measures, it becomes our duty to be prepared to operate, after the risks of operation have been considered by all who are interested in the result. And even then my experience justifies me in stating that in all but the exceptional cases the cutting or other forcible measures should always be delayed until the conservative plan had been tried and its failure to meet the indications has been demonstrated.

The natural inclination of each femur is toward the median antero-posterior plane of the body. Each bone forms with the perpendicular an angle of 15°. The plane of the knee-joint is such that it adapts itself to the maintenance of the erect posture, and this plane is mainly secured by a difference in the condyles of the femur. If we place the femur perpendicularly before us, we see at once the difference between the condyles, the inner being, according to Holden, one half inch lower than the outer in the adult. On the other hand, if we examine the articulating surface of the head of the tibia, we find that its general horizontal plane is at right angles with the vertical axis of the shaft. So that, while the direction of the femur is oblique, the principal modification required to insure perfect adaption is made at the condyloid surface.

In the condition known as knock-knee, there is always some change affecting the condyloid plane which results in an altered relation of the tibia to the femur. The bones join each other at the knee-joint, and the articulation is practically a double one, that is, there are two condyles articulating with two separate surfaces on the head of the tibia. Any process which results in an atrophy or a hypertrophy of either of the condyles or produces a deepening of one of the surfaces with which they articulate, must change the relation of the bones to each other and produce a lateral deformity. And if there should exist an hypertrophy of the outer condyle or a deepening of the inner facet on the head of the tibia, the astragalar end of the tibia would be thrown toward the median antero-posterior plane of the body, producing an angular sort of bowleg or the reverse of knock-knee. Or, if there were an atrophy of the inner condyle, or a hypertrophy at the outer facet, the result would be the same. On the other hand, if there were hypertrophy of the inner condyle or atrophy of the outer condyle, or atrophy of the outer facet, or hypertrophy of the inner, we would have the position known as knock-knee. In addition to this, we must add abnormal conditions of the shaft of the femur above and the tibia below. For, if the normal direction of either bone is lost laterally, a condition of genu valgum or genu varum would ensue as a matter of course. But in knock-knee proper the essential changes take place in or near the epiphysis, with little or no change in the shaft of the bones. And we would make this distinction between the conditions which we are considering:

1st. Cases involving a marked degree of curvature of the shaft of the bones above or below the knee-joint are recognized as bowlegs, with secondary malposition of the joint, but without any essential alteration of the transverse condyloid plane.

2d. Cases involving the joint itself primarily and producing secondary changes which alter the direction of these long bones, but without any marked curve, are almost invariably cases of knock-knee; for, while we have both genu varum and genu valgum, the former involves in all but the exceptional cases a *deformity* in the shaft of the bones only, while the changes seen in knock-knee are frequently unassociated with marked changes or curvature in the diaphysis; and still further, the changes in knock-knee, other than those found in the condyles, are almost always seen, not in the tibia below, but in the femur above; while, as is well known, in bowlegs we frequently have a curvature of the tibia alone, the removal of which curvature results in the restoration of the normal direction of the limb. There is one constant and pathognomonic feature of knock-knee, and that is, the internal condyle is always found to be comparatively larger than the external, or, as McEwen states it, ''an alteration is found in the plane of the condyles,'' so that the whole internal condyle is larger and the external smaller, speaking comparatively. This prominence of the internal condyle may be due to an increase in size at the diaphysis of the femur, just above the epiphyseal line. In the majority of the cases I have seen, this prominence of the internal condyle, apparent or real, has been marked, and though, I have no doubt, cases present where the alteration of the condyloid plane may be partly due to an inward bending of the femur (McEwen claims that this is a very common factor), it is quite certain that a restoration of the condyloid plane removes all the essential features of the deformity. McEwen also states that there are cases where the deformity is due to an intrinsic lengthening of the internal condyle, and that there are usually two or three factors which enter into the formation of the knock-knee. We cannot avoid the conclusion, however, that these factors, whatever may be their apparent value, have their cause in a primary change, whatever it may be, which affects the process of nutrition at the femoral epiphysis, or in the shaft immediately above it.

I wish I could satisfactorily answer the query which here presents and say what is the cause of this disturbance of nutrition. Is it a process of malnutrition affecting the system generally, but finding its principal expression at the knee-joint? Can we make the generally accepted definition of rickets apply to all the cases that are found in these localized expressions of osseous deformity seen in knock-knee? If so, why is it so localized? For knock-knee frequently presents as a strictly-speaking unilateral deformity, and affects only one bone or one articulation. Rickets is a constitutional disease, and is characterized by some conditions which are not always found in knock-knee. In many cases, also, there are, or have been no symptoms except the slowly progressive deformity. Rickets is a constitutional condition, which may be followed by various osseous deformities. But we cannot overlook, if we make careful inquiry into the history, the symptoms of the constitutional condition when it is found in these cases.

There is, certainly, something which remains to be explained regarding the etiology of knock-knee; a something which time and patient investigation only can develop. Why, for example, should we find that the epiphyseal changes, which are known to exist in joint-disease, produce such marked symptoms, muscular, neural, and otherwise, when in the atrophic or hypertrophic changes involving these same structures in knock-knee there should be no such symptoms? What peculiar process can there be affecting the nutrition of the epiphyses of a non-inflammatory character and having a local cause? What are the changes which produce either atrophy or hypertrophy in other parts of the body? If we apply the answers which must be made to these questions to the conditions found in the knee-joint in genu valgum, we are led far away from the ordinarily accepted definition of rachitis. And there are no greater changes found in knock-knee than are often seen in the many localized expressions of disturbed nerve-centers or localized disturbances of the vascular system. At any rate, there is something more than simple "malnutrition" in these cases. There is something beyond these purely objective signs which are found in and which accompany these crooked joints. What it is I do not know. If we recognize that these changes, whether inflammatory or trophic, are not always associated with rachitis, we have, I firmly believe, taken a step in the right direction.

Knock-knee is defined by McEwen as being a condition of the lower extremity in which the center of gravity passes to the outside of the knee-joint. This is undoubtedly a description of the mechanical relation of the parts. But there is a condition where the line of gravity is thus displaced when the patient stands which does not exist when the patient lies down. We may have knock-knee due to ligamentous relaxation. I have seen many cases of this type produced in children by the careless application of traction through the unprotected knee-joint, in the mechanical treatment of hip-disease. But I never knew a case of this kind to develop the characteristic knock-knee with articular bony changes or to result in a rigid lateral deformity. If we were to look for a local cause for knock-knee, we would be likely to attribute it to a primary relaxation of the ligaments. For, when the ligaments are relaxed, the inward slant of the femur throws the knee-joint toward the antero-posterior mesial plane when the patient walks, and the weight of the body then falls on the outer condyle. If the epiphyses are in a normal condition, and trophic changes have not occurred, and there is no central or general cause at work to produce it, knock-knee is no more likely to develop in these cases than is a permanent lateral curvature of the spine from unequal length of the lower extremities. In the one case we find a malposition of the knee-joint, which is absent when the patient removes all weight from the joint, and in the other we find a symptomatic lateral curvature which also is wholly removed when the patient lies down. Ligamentous relaxation, *plus* the predisposing cause, whatever it may be, is very sure to result in true progressive genu valgum. Ligamentous weakness, *minus* the predisposing cause, corrects itself in time if simple protection be afforded. But do not confound the pseudo-form with true knock-knee. We should watch, and, if necessary, treat this ligamentous weakness of the knee, but not record it as a case of knock-knee and report a cure after three or four months' treatment. We can, therefore, divide knock-knee into two classes: first, true genu valgum, where there are actual changes in the transverse plane of the joint, with or without osseous curves above or below the articulation, and secondly, lateral malposition of the joint, due wholly to ligamentous relaxation, which malposition, owing to the direction of the femur, always presents a knock-knee in locomotion, but which is, as a rule, unassociated with any femoral or tibial changes. We will briefly consider these two conditions and point out the means of treatment in each class of cases.

In true genu valgum we are met with some problems in the way of treatment which demand much thought and careful consideration before we decide as to the best course to be pursued. If we see the deformity in the early stage, there should be no doubt about the means to be pursued or the result. But if the deformity has existed a long time, and there is a very considerable change in the articular surfaces, there is a doubt which can only be settled by years of future observation. Mechanically speaking we must attempt to accomplish in knock-knee just that which we attempt to avoid in the treatment of chronic joint disease, namely inter-articular pressure. We must make a fulcrum of the articular surface of the inner condyle in bringing the tibia into its normal position.

Why is it that we cannot reduce this lateral malposition easily in knock-knee? I have often asked myself this question. If the changes in the transverse plane are due solely to the cause assigned them, why can we not by simple manual pressure restore the limb to its normal position by creating a gap at the outer condyloid articulation? Is it due to a contracted state of the lateral ligaments from position or to retraction of the biceps or to both? If so, why not simply divide the resisting tissues and at once restore the limb and maintain it there? But division of these tissues does not suffice, and attempts in this direction in the hands of able men have not been successful. Are the changes which are discovered *post mortem* at the external glenoid cavity of the head of the tibia primary or secondary? There are problems in the etiology and pathology of knock-knee that have not been even touched yet, and while it is all very easy to say "it is nothing but rickets," I am sure that there is a something about these cases that rickets does not explain. But we have a lateral resistance to overcome, and be its cause what it may, and the question is, Shall we attempt to overcome it and trust to time and to nature to accomplish the rest, or shall we adopt some of the many operative procedures that are being tried in various parts of the world? Here again let me say, I do not think of operating until I have exhausted every known resource that mechanics give me, and that case will indeed be a confirmed one which does not yield to the mechanical means at our disposal. In the simpler cases it cannot fail. In the worst class of cases, if time and attention be given, it ought not to fail, unless the vulnerability of the superficial tissues is so great that the requisite pressure cannot be tolerated, even when applied in the manner I shall shortly describe. But first let me briefly allude to the various operative measures that have been recently introduced. The first is *redressment force*, an operation that consists of making a fulcrum of the internal condyle and the application of sufficient force to produce an *immediate* restoration of the crooked limb. It is, in my opinion, a very unsafe proceeding. It has produced death, and is very apt to be followed by serious inflammation and an unstable articulation. I cannot imagine a case which would justify its use. Another plan is Ogston's. It consists of a subcutaneous section with a saw of the prominent internal condyle, which, when separated, slips upward and allows of an immediate adjustment of the tibia in its normal position. But the joint is opened. The measure has been condemned by eminent men, and, like Delore's method, first mentioned, has produced death and unsatisfactory results. Reeve's method, a modified Ogston's operation, consists of little more than the substitution of a chisel for the saw. It is preferable to Ogston's, but presents many of its objectionable features, though if care be used the joint is not opened. Indeed all these and other operations which involve opening the joint, or the danger of doing injury to it, or a separation at the epiphyseal line, are, in my opinion, unjustifiable procedures, and they would still be so were there no immediate danger to life or joint, inasmuch as we do not know, and cannot, for years to come, estimate the effect of these bony epiphyseal sections or the *redressment force* upon the future nutrition of the epiphyses themselves and through them upon the development of the femur or tibia. We do not know as yet the ultimate result of these operations. The immediate results in many instances have proved satisfactory. If it should ultimately be demonstrated that there is no interference with nutrition causing an arrest of development, or no hypertrophy from the more active nutritive process brought about by the reparative action, we would have ample grounds upon which to judge of their merits. But there is another procedure which does not involve the joint. It is McEwen's operation. It is performed by making an incision on the inner side of the thigh, just above the condyles, and cutting through the bone with a chisel—the "supra-condyloid operation." This procedure possesses the merit, at least, of non-interference with either the joint or the epiphysis, though it is open to the objection that, while bringing the limb straight at once, it does so by carrying with it the unmodified and abnormal transverse plane. Still there can be no question, I think, about the choice of operations if one must operate. I should choose McEwen's at once as being the one least likely to be followed by embarrassing ultimate results.

There are other operations to which I need not call attention on this occasion; they are all more or less attended with open wounds, though they are called subcutaneous. Listerism has been relied upon by all these operators, though many have been disappointed by the appearance at times of suppuration and its attendant evils.

The method which I use is a simple one, and though its capabilities have never been thoroughly tested it has been attended with results already which warrant its extensive trial. As before remarked, the principle involved in its use is the concentration of inter-articular pressure upon the prominent internal condyle. If we cannot produce absorption at this point, we can at least make it the fulcrum by which we stretch the opposing tissue at the outer aspect of the articulation. The degree of force we can apply is very great. But we must not err and make the application of this force continuous. The fact that the superficial tissues will bear a greatly exaggerated pressure for a few seconds or minutes without harm in the mechanical treatment of such deformities as knock-knee, bowlegs, club-foot, etc., is of great service to us. I have often illustrated this fact in the application of forcible momentary traction in the treatment of club-foot. And I have also found that what was gained by this momentary exaggerated pressure was not lost by a few moments or even a few hours of rest. I apply the same principle to the mechanical treatment of knock-knee. And there are many conditions to which this same principle is applicable. The momentary toleration of severe pressure is recognized in acute surgery, and its value as applied to the reduction of chronic deformities is very great. It opens a large field and solves many problems which before were looked upon as being practically beyond solution. It brings the treatment of many deformities heretofore considered as incurable without the knife within the range of strictly-speaking mechanical therapeutics.

The instrument by which we apply this exaggerated force in knock-knee (Figs. 1 and 2) consists of two parts joined together by a lateral hinge at a point which corresponds with the most prominent part of the internal condyle. The upper or femoral part has two straight pieces of bar steel, held together above by the conventional band.

The inner lateral bar terminates below in the upper part of the hinge. The outer bar is not continuous with the calf bar, but is made to terminate about 1½ inches above the knee-joint, where another band joins it to the inner

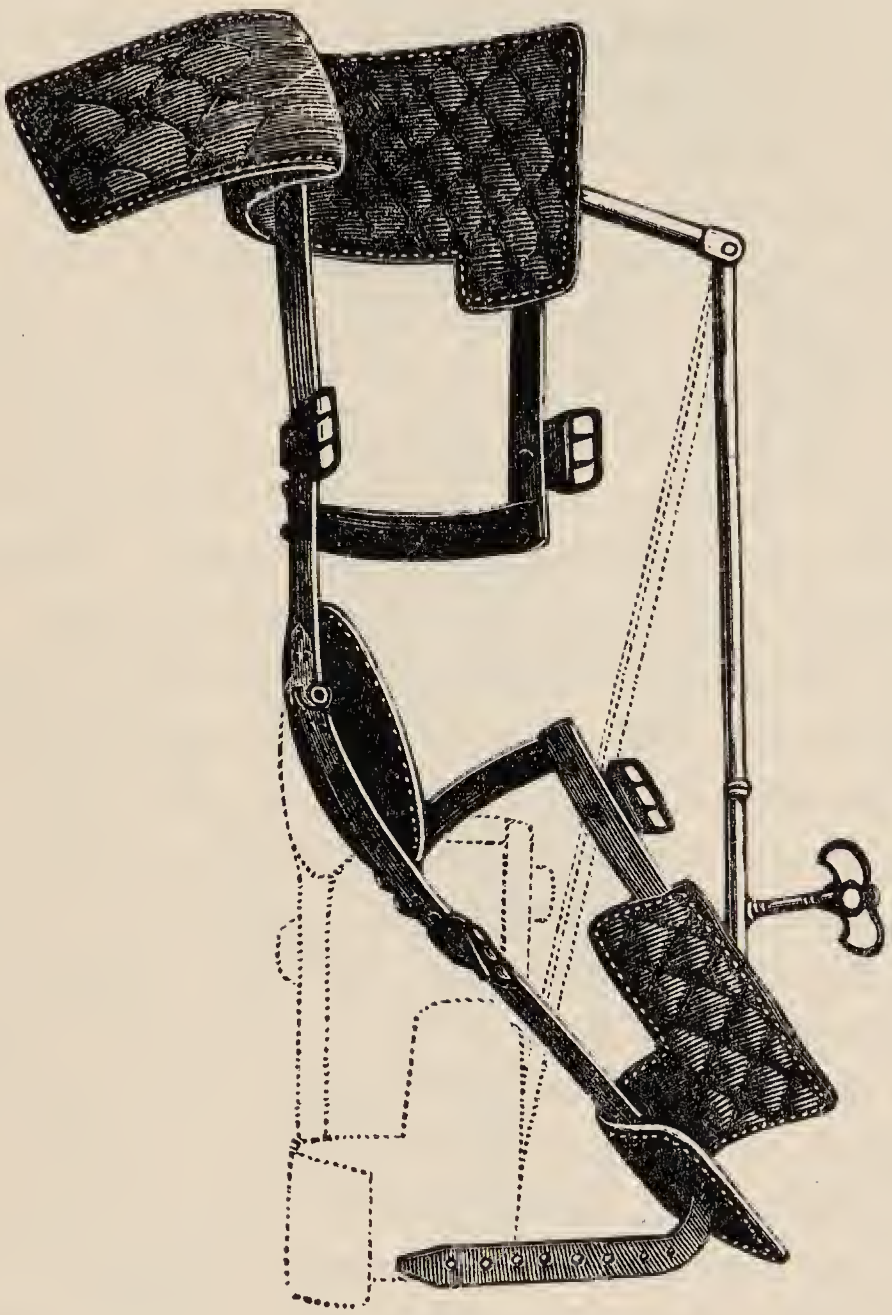

FIG. 1

bar. The lower part is much the same as the upper, that is, the inner bar assists in forming the hinge on the inside at the condyle, and the outer bar terminates 1½ inches below the joint. The lower bars are joined by two bands, one at the distal ends, the other at the termination of the outer bar just below the joint. On the outer bar of the thigh-piece is placed at a proper angle an extension rod which is inserted in the outer bar of the calf-piece near its lower end. We have thus practically a knee splint which acts in a purely lateral way and much the same as the ordinary knee splints found in the shops act to extend the knee-joint antero-posteriorly. The center of motion is at the inner condyle, and the force which is applied acts directly to restore the lateral malposition. There is placed under this hinge over the inner condyle a soft pad, and in order to prevent the limb from rotating, a few strips of adhesive plaster are applied. When the instrument is adjusted, as in club foot or indeed as in other chronic deformity, it is made to fit by means of the lateral extension rod *the exact position of the deformity.* We now apply any desirable amount of pressure with the key. We see the limb restrained in every other direction pass toward the normal one, and we hold it after applying the exaggerated pressure for fifteen, thirty, or even sixty seconds. We then remove all force and instruct the mother or nurse in the application of the pressure. All severe pressure is removed at night. During the day this exaggerated force is applied every half-hour or hour. The only thing to be feared—and this has never happened—is a separation at the femoral or tibial epiphyseal line—a result which is sought for by many. But you need not apply force enough to incur any serious risk: you can in all but the very exceptional cases bring the limb into position after a few weeks. In these exceptional cases, use McEwen's supracondyloid operation as a last resort only.

After the limb is straight, we must simply put on some effective retention splint with antero-posterior motion at the knee-joint. Time and nature will do the rest, and we will succeed in accomplishing an *ultimate* result which will,

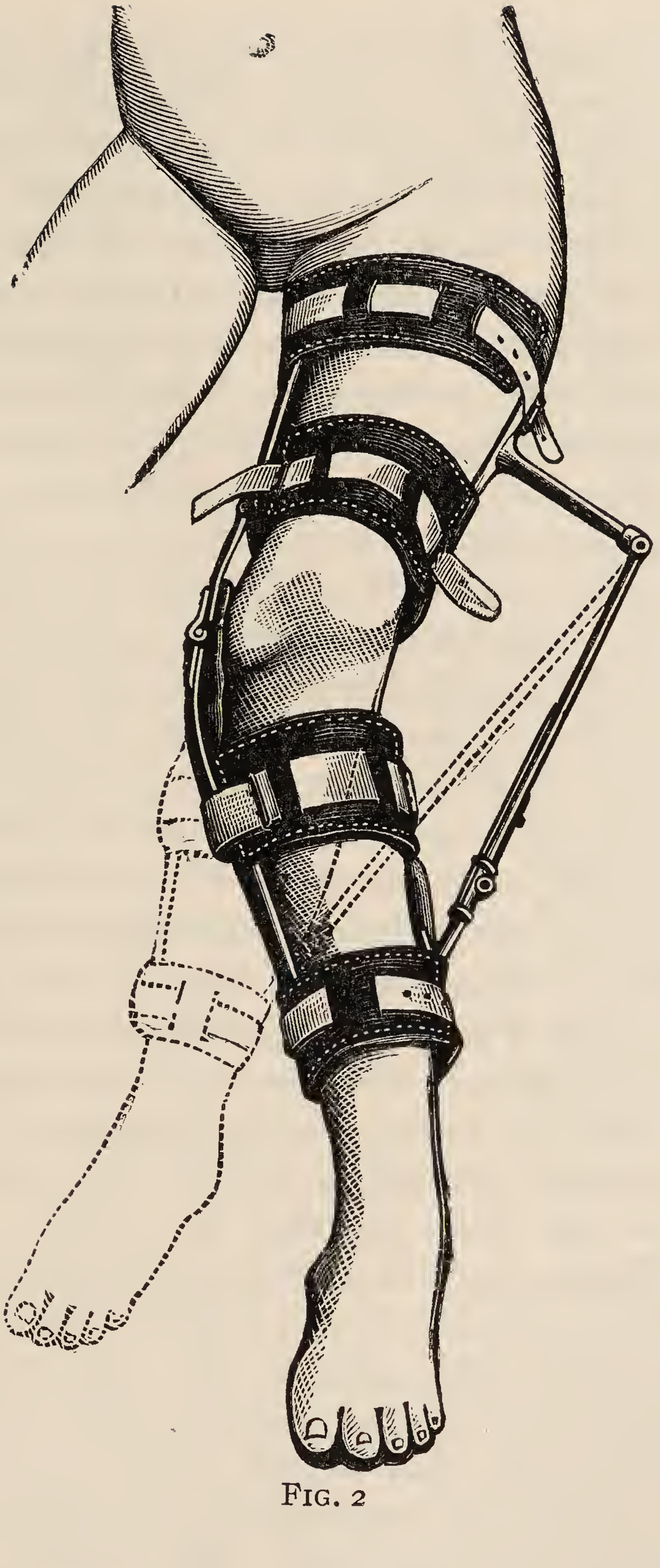

FIG. 2

I doubt not, equal, if it does not excel that obtained by any of the operations to which I have referred. I have reason to believe that if all pressure is removed from the atrophied outer condyle, osseous deposit will occur. In one case which I treated in St. Luke's Hospital, this deposit did take place rapidly. We certainly would look for a better result than would follow the epiphyseal sections, and in young children especially, where a very considerable growth of the femur and tibia is yet to take place, the method I have described is much preferable, in my opinion, to any operation which involves the use of the knife, the saw, or the chisel. In adolescents the difficulties are increased as to mechanical treatment and are lessened so far as interference with growth is concerned. But with my present experience I should not hesitate to try the mechanical method in even the most confirmed adolescent cases.

We will now pass to a consideration of bowlegs or genu varum. In this condition we find an outward curvature of the femur or tibia or both. Speaking generally, it may be described as that condition where a line drawn from the head of the femur to the ankle passes inside of the knee-joint (McEwen). The amount of deformity will vary greatly in different cases. The curvature may be so slight as to be scarcely perceptible, or the femur above and the tibia below may form together the segment of a circle; but in all these cases the curvature of the bones is the patent factor, and the transverse plane of the joint is, in children at least, little affected. In many cases also, as before stated, the curvature is in the tibia alone, and it is very rarely the case that we find a unilateral deformity in bowlegs—another fact which shows the difference between this condition and knock-knee. Genu varum on one side and genu valgum on the other is sometimes observed, though not frequently. In the vast majority of cases, the curve is bilateral, with perhaps a slight femoral, but a very apparent tibial bending.

In seeking a cause for bowlegs, we must look for a condition which affects the diaphyses rather than the epiphyses, for one will frequently see cases of bowlegs without any apparent epiphyseal changes at the knee-joint, or if there is any change it affects the entire epiphysis, not its inner or outer half.

On the other hand, it certainly rarely happens that we find knock-knee without the unilateral condyloid changes that have been pointed out. And rickets or the condition which has been described as such, and which is accompanied by enlarged and otherwise altered epiphyses in other parts of the body, and especially at the distal ends of the radius and ulna, is almost always found to be associated with bowlegs. And so also are the other generally recognized signs of rachitis found in bowlegs. These signs are certainly seen less frequently in knock-knee. If the same general cause operates to produce both these conditions of genu varum and genu valgum, it certainly varies greatly in its manifestations. So great is this difference that I cannot doubt that a different etiology exists, or at least that something more than has yet been discovered is acting as a factor in the production of the true knock-knee.

True bowlegs is undoubtedly of rachitic origin, as before remarked, and the *deformity* lies in the diaphyses. But in bowlegs as well as in knock-knee there frequently exists a relaxation of the knee-joint ligaments which gives an apparent curve to the femur above and which makes the deformity appear much greater than it really is. Children thus affected will walk very badly when unsupported, owing to this ligamentous weakness, and as a consequence the knee-joint is thrown out much further than normal, from the true line of gravity in the act of walking. This in itself has a marked tendency to increase the leverage, if I may so speak, upon the curved tibia, and if it is softened the progress of the bowing outward is rapid. We met with many cases of bowlegs, however, where there is no abnormal lateral mobility at the knee-joint, and these are the cases which are most difficult to relieve. Sometimes the bones will remain soft for a long time, in others the bones will rapidly harden in their deformed position. We all know that bowlegs may recover spontaneously. I have seen several bad cases in children over five years old straighten without treatment. Still we cannot afford to take any risks and we have no means as yet by which we can decide which cases will recover without treatment. The greater number of cases which are due to ligamentous weakness alone undoubtedly recover spontaneously, unless the rachitic tibia exists. If there is ligamentous relaxation without any curve in the tibia whatever, we can afford to watch the case for a brief time without giving any mechanical support. But the abnormal lateral mobility at the joint *plus* the abnormal curve in the tibia demands active treatment at once.

The mechanical treatment for bowlegs which is described in works on surgery is entirely inadequate to meet the indications. It may briefly be described as the application of retention splints to the position which is already a deformed one, but which in effect is only a support to the limbs in their malposition. There is nothing which acts progressively to overcome the curvature itself, and mush valuable time is lost in using them. All indeed which the ordinary "bowleg braces" accomplish is to aid in sustaining the weight of the body when the weight falls upon the curved bones. They usually do not accomplish this effectually unless they are modified almost daily, for the weight of the body in the majority of cases will so affect the lateral uprights and the horizontal bands that what is an accurate support to-day will become an inefficient one to-morrow. At night when the patient is lying down nothing is done, while at this time we can apply an

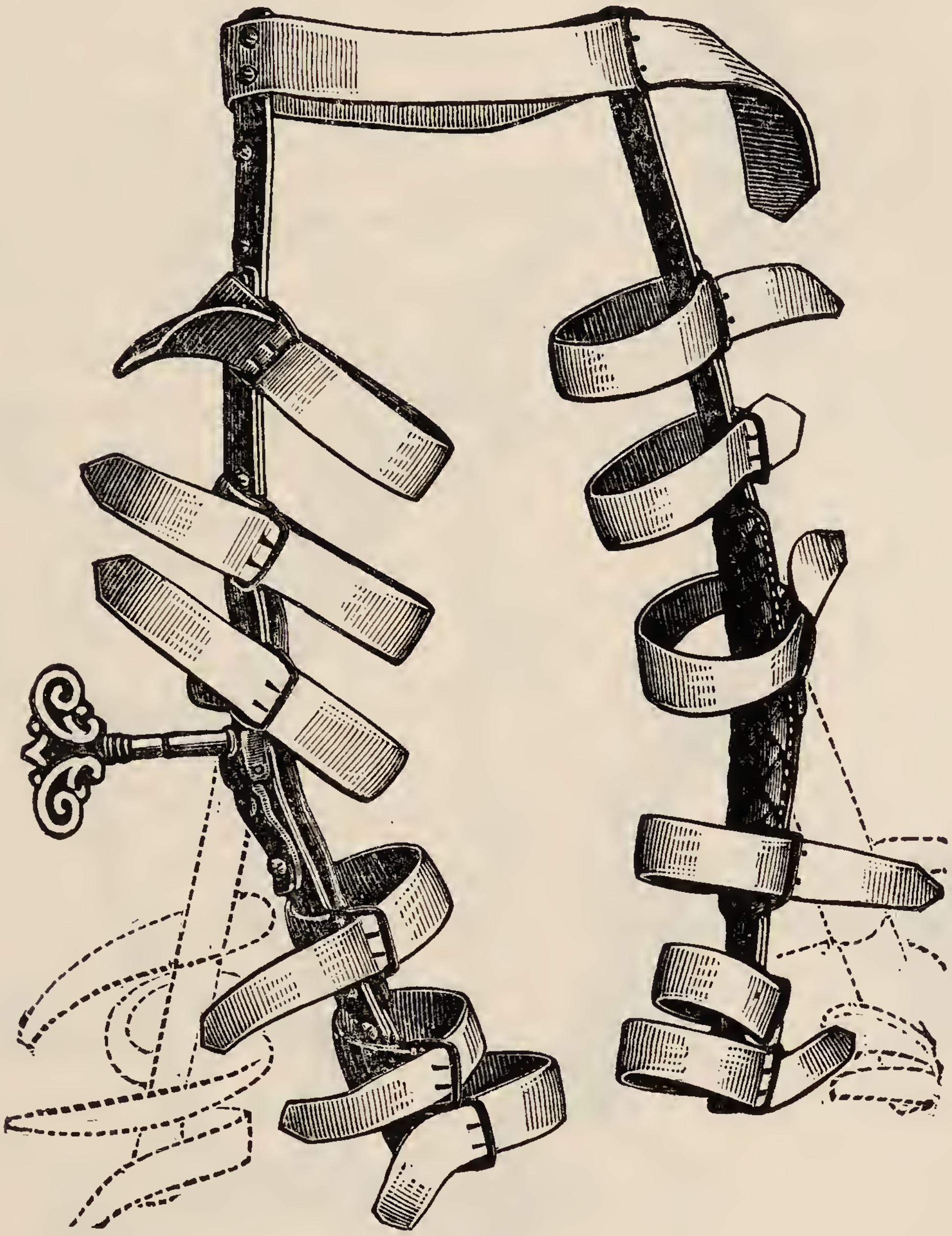

Fig. 3

efficient support which does not have to sustain the weight of the body before it affects the curvature.

My plan is as follows:—During the night and at certain hours during the day I apply an instrument which consists of two lateral bars (see Figs. 3 and 4) passing from the trochanter major to the sole of the foot without any joint save one which has lateral movement, and this is introduced at the point of greatest convexity of the tibia. These lateral straight bars are joined together at the pelvis

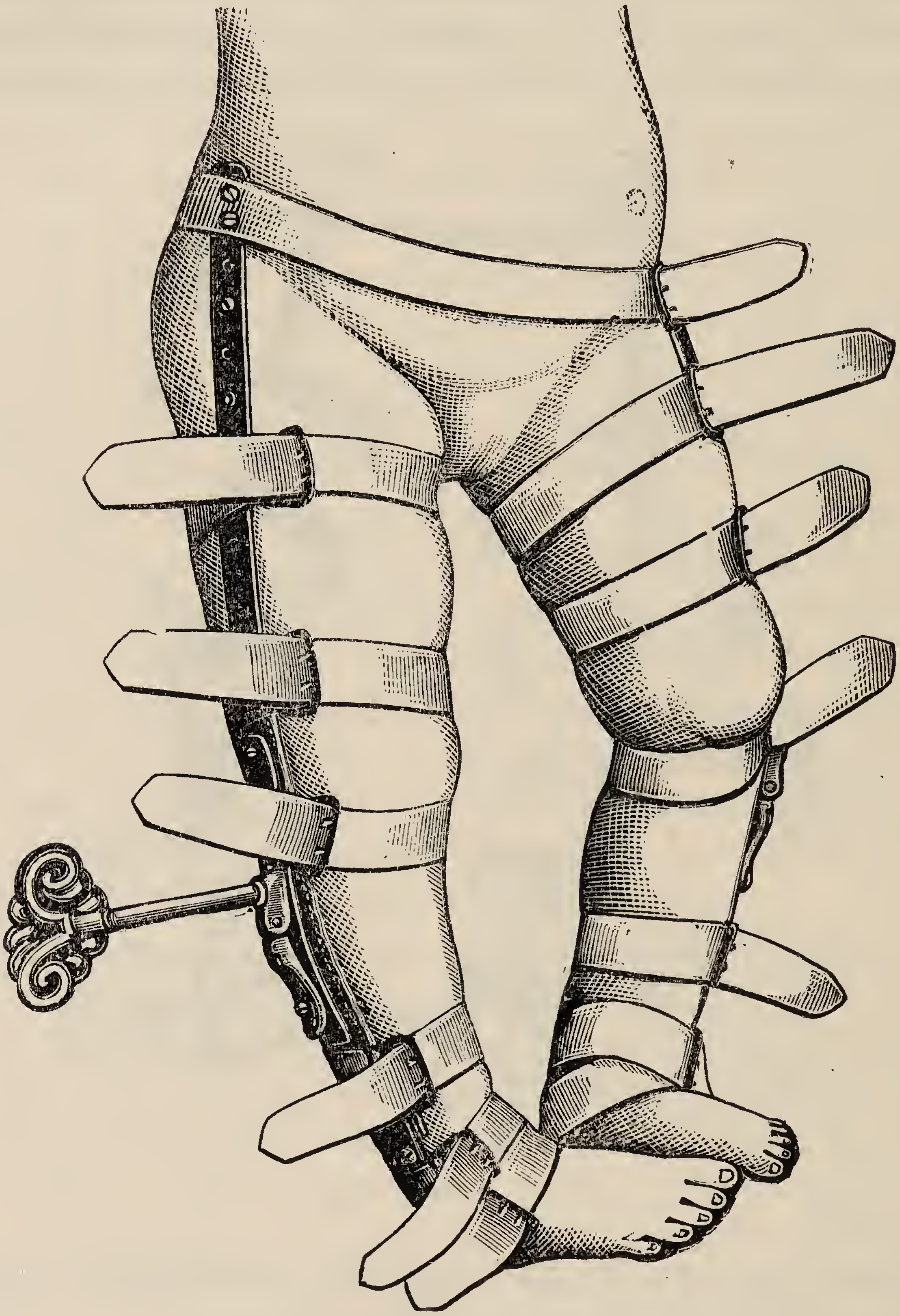

Fig. 4

by a webbing band. They are securely, not tightly, bound to the limb at various points, also by webbing bands, and especially above and below the knee, and at several points below the lateral joint in the instrument. This lateral joint is constructed with a hinged lever and screw, just as in my lateral club-foot shoe. In the latter we *push* the tarsus *in;* in the former we use it to *pull* the curved portion of the tibia *out.* The instrument is adjusted to *exactly fit the deformity.* We then fasten the webbing straps securely, and especially those which include the malleolus and the astragalus. You know there is no lateral movement at the ankle-joint, and we can therefore regard the astragalus and practically the os calcis as being part of the tibia. When the straps are fastened, we can apply with a key all the force required. We can keep up a moderate degree of pressure all night. In the morning, and if necessary during the day, we can again apply, at such intervals as may be deemed expedient, the principle of momentarily exaggerated pressure. These instruments are not annoying or irritating, and in one case in which the child was very nervous and timid, the mother applied them and exerted all the necessary pressure after the child was asleep without awakening the patient. After the night instruments are removed, the child is placed in a "day splint" which is made like the conventional bowleg retention splints. These day splints are worn whenever the patient walks, and the parents are instructed to bend the horizontal bars occasionally so that, when adjusted, the outer lateral bar fits the outline of the limb closely. In these, as indeed in all orthopædic cases, one must spend all the time necessary to instruct the parent or nurse in the detail of the work. We will have, of course, to see the patient occasionally and to make modifications as indicated. In the great majority of cases this treatment is all that is required to remove the deformity. Many cases that are now condemned to operative surgery can be cured by this method. Just how far it will answer in the older and more stubborn cases I do not know. I have treated thirteen cases, the oldest seven years, by this method, and all of them thus far with excellent results.

But operative surgery has lately included bowlegs in its list, and there are several procedures which are used by surgeons to accomplish the reduction of the deformity. Prominent among these are the section of the bones by saw or chisel, and fracture by instruments which have been devised for this purpose. There is no "condylotomy" for bowlegs. The operations are all directed toward the curved tibia and sometimes to the femur; I need not speak of these operations in detail. I will simply say that if I were called upon to use any method other than that I have described, I would use the osteoclast. I see no need of making a compound fracture when we have at hand the means of producing a simple one.

ON INDISCRIMINATE CIRCUMCISION[1]

A FEW years ago the ancient rite of circumcision was brought to the notice of the profession as a remedy for certain ills of childhood. Since that time no one surgical operation has been performed, in all probability, more frequently. First brought to the attention of the profession by Dr. Sayre for the relief of "reflex paralysis" dependent upon genital irritation, it has come to be almost a habit with some surgeons to refer to phymosis with or without adherent prepuce a long list of evils. The result has been that many times this operation has been performed when no necessity existed for its performance, and many parents have been led to expect great and permanent benefit when no benefit accrued.

Like many other "advances" in surgery, this simple procedure was accepted by a great number of the profession upon the basis of assertion rather than that of demonstration. The premises were assumed, and the published results, apparently, satisfactory. Whether the patient suffered from "reflex paralysis" and his condition was one of induced "spinal anæmia," or whether there were obscure symptoms which were, in reality, those of incipient joint or spinal disease, or if a child were microcephalus and lacked co-ordinating power, or, indeed, if any unexplained or anomalous symptoms presented, the one great remedy with many has been circumcision.

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[1] *Annals of Anatomy and Surgery*, May, 1881.

It has been my fortune to see many cases where this operation has been performed. Indeed, it is not an infrequent thing to find, in those patients who have been for some time under the care of other surgeons, that circumcision is an accomplished fact. I could furnish quite a number of cases where this procedure has been adopted for the cure of what has proved to be incipient hip disease. There are those who have been informed that adherent prepuce caused spinal disease. Indeed, circumcision has been performed for the cure of congenital club-foot! And a long array of cases present where not only circumcision but clitoridectomy has been performed for what has been called "reflex paralysis," when the condition for which relief was sought was an organic lesion of a great nerve center.

I have never seen a case of reflex paralysis, in a child, from genital irritation. My friend, Dr. E. C. Seguin, informs me that his experience is like my own in this respect. I have seen several cases which have been diagnosticated as such, but upon examination they proved to be instances of lateral sclerosis of the cord. In these cases where circumcision has been performed, the operation, as a matter of course, produced no more effect than would the same measure upon the condition recognized as infantile paralysis. The great disappointment expressed by the parents of these unfortunates, is a fitting comment on the uselessness of this operation as a remedial measure for other than simple local or directly traceable genito-urinary troubles.

And yet this operation has sometimes been followed by temporary relief in cases other than genito-urinary. The following case will illustrate this fact:

In 1879 a gentleman residing in New Jersey brought his son to me with a history which gave symptoms that had existed for a few weeks. These symptoms found expression principally in the neck. The child—3 years old—was unable to properly rotate the head, and motion was more or less restricted in all directions. There was also some occipital pain and distress upon any sudden motion of the head. These symptoms had been considered by the local surgeon to be of reflex origin, and due to phymosis and adherent prepuce. The parents were induced to have circumcision performed, and a day or two after the operation the pain became less and the head movements were nearly normal. The operating surgeon was jubilant, inasmuch as another surgeon had pronounced the lesion to be one of cervical spondylitis. Some doubt, however, existing in the minds of the parents, owing to these conflicting opinions, the child was brought to this city, and Dr. H. B. Sands was consulted. Dr. Sands made a diagnosis of cervical spondylitis, and kindly referred the patient to me. The parents, after the relief which followed the operation and the assurances of their own surgeon, accepted the diagnosis of Pott's disease with some reluctance. They asked me pointedly how I accounted for the relief which the operation, apparently, afforded. My answer was as follows: That the relief did not come at all from the operation, but from the enforced rest which it and the subsequent treatment necessitated. This was so without any doubt; for in a few days, after running about while waiting for the apparatus, the symptoms returned, and were much more severe than before. It is now over two years since the child first came under my observation. He is still under treatment, though nearly well.

Not long since a patient of mine who had incipient Pott's disease, but who was waiting for her support, fell and severely sprained her ankle. For a week she was obliged to remain very quietly upon her bed. At the expiration of the week it was noticed by all her friends that she had very greatly improved in her general condition, and the spinal symptoms were much relieved. The enforced rest in this instance, as in the one where circumcision was performed, produced a very decided temporary improvement. It plainly indicates the nature of the treatment to be pursued in similar cases. I have seen several cases, which proved to be incipient spinal disease, where the enforced rest of circumcision produced a more or less marked temporary improvement. But the attending surgeons, not having recognized the disease, made the error of attributing to the effect of the operation the benefit which the recumbent position produced.

I might relate other cases illustrating the effect produced in incipient and obscure joint and spinal disease by the temporary rest following circumcision, in which the operation had been performed for the relief of "reflex symptoms." In some instances, also, the operation has been performed at a time when, as frequently happens in the early stage of chronic joint lesions, a remission of the symptoms occurred. But the fact that temporary rest for a few days produces oftentimes a marked but deceptive relief, should always be appreciated by the surgeon, not only as a matter of diagnosis, but also as an indication for early and prompt measures to secure rest in a permanent form.

But in the very great majority of instances which have fallen under my observation, where the prepuce has been removed for the obscure symptoms which usher in the first apparent stage of chronic joint or spinal lesions, the operation, as may well be inferred, produced no effect whatever upon the supposed reflex symptoms. The patient derived the ofttimes questionable benefit of an uncovered glans penis; but the operation was not performed, in these cases, with this end alone in view.

The class of cases to which this operation is supposed to be especially applicable is "reflex paralysis" of the lower extremities. Among the many cases of this so-called "reflex paralysis," that I have seen, I have found none that did not prove to be a lateral sclerosis of the cord, with a greater or less modification of co-ordinating power. In some instances the lack of co-ordination would be so slight that under ordinary circumstances no deviation from the normal would be noticed. Some movements would be awkwardly executed, especially those developed by running or jumping; or there would be a slight ataxic movement of one or both of the lower extremities in the ordinary movements. Still there would be a good general nutritive condition with or without some impairment of the mental faculties. I have several such cases now under observation.

One, a boy of about three years, of generally good nutrition and fair health commenced to walk at twenty months. His gait was awkward, but there was no deformity. There was neither atrophy of the lower extremities nor apparent contraction of the muscles. Lying down he could use his limbs as well as any child of his age; and it was only when rapid locomotion was attempted that the unnatural gait became markedly noticeable. There was no trouble with the upper extremities—no especial lack of mental development; it was simply an awkward gait—and as such it was regarded. It was looked upon as a case of "reflex paralysis" by the family physician, and was so treated. Ordinary remedies having failed, it was decided to circumcise. This was done; but the expected relief did not follow. The patient became progressively worse; and at the time I first saw him, he walked with a decided inversion of the right foot, and there was slight contraction of the gastrocnemius, the tibialis anticus and the tibialis posticus. These contractions yielded to gentle and persistent manual pressure—but the deformity returned almost instantly after the pressure was removed. There was also greatly exaggerated tendon reflex, especially on the right side. The other reflexes were also active, and the child gave a history of early convulsions, and a nocturnal cry very similar to that noted in the osteitic forms of joint disease and in acute hydrocephalus. He was also "very nervous."

The diagnosis was lateral sclerosis of the cord (Seguin's tetanoid paraplegia[1]), and the prognosis was far more grave than the parents had been led to suppose.

I have recently seen two cases of "reflex paraplegia" in girls—now 6 and 7 years of age, respectively. As in the preceding case, the lesion was lateral sclerosis, and in each clitoridectomy had been performed for the cure of supposed reflex symptoms. It is now four years since these operations were performed, and in both instances the parents were assured that complete relief would follow! I might further duplicate these histories and relate some remarkable experiences, but I forbear. True reflex paralysis, depending on general irritation, is exceedingly rare. Lateral sclerosis in childhood is not rare. Occurring in infants, often if not always congenital, frequently unnoticed until delayed locomotion or an irregular gait calls attention to it, and being unaccompanied by pain, the lesion is often supposed to be reflex paralysis, and is treated in the manner described. The assumption that a coincidental genital irritation has anything to do with this state of affairs is too absurd for contemplation.

I wish to earnestly protest against this habit of indiscriminate circumcision. In certain cases, where there is undoubted preputial irritation, the indications for this operation are plain and unmistakable, or when, for sanitary or other reasons, circumcision is deemed advisable, there cannot be any objection to it. On the contrary, much good frequently results, and many patients have derived permanent relief from incontinence of urine, frequent priapism, etc. But there can be no doubt whatever that in many cases of organic disease, where the symptoms were very insidious, and hence misinterpreted, the operation has been advised and executed as a means of cure where there was no connection whatever between the so-called reflex symptoms and the frequently inferred genital irritation.

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[1] "Description of a peculiar paraplegiform affection." By E. C. Seguin, M. D.—*Archives of Scientific and Practical Medicine*, Feb., 1873, N. Y.; and

"The present aspect of the question of tetanoid paraplegia." By E. C. Seguin, M. D.—*Archives of Medicine*, Feb., 1879; N. Y.

It does not come within the scope of a brief note like this to describe in detail the symptoms of lateral sclerosis in children; nor can I do more, in this connection, than to call attention to what has been said upon the insidious invasion of chronic lesions by various writers. The condition which has so often been called "reflex paralysis" is recognized by different authors as Erb's paralysis, spastic paralysis, postero-lateral sclerosis, and tetanoid paraplegia. Those interested in the subject of lateral sclerosis will find much valuable information and extensive bibliographical references in Dr. Seguin's papers already referred to.—*Editorial.*

THE HYSTERICAL ELEMENT IN ORTHOPÆDIC SURGERY

Read before the New York Neurological Society, December 1st, 1879

THERE are two important systems involved in the process of acute local inflammation, namely, the vascular and the nervous. The pathological changes wrought through the operations of the one and the contributive influence of the other in forming the group of symptoms which characterize this condition, need no comment. A typical, local, acute inflammation cannot easily be mistaken for any other pathological state. As we descend in the scale and pass to a study of the sub-acute local lesions, we find that the difficulties in the way of diagnosing this condition are not materially increased. But in the first stage of certain chronic diseases we lose many of the symptoms by which the local, as well as the general, evidences of inflammation are recognized and, aside from the length of time which the history of the case may have covered, there are, not infrequently, confusing elements introduced into the etiological and semeiological factors which demand more attention, I think, than has been accorded to them by some of our most eminent teachers.

It would seem that the symptoms that arise from pathological changes, which, whatever be their etiology, affect principally the local, vascular tissues of a part could be easily distinguished from those which find expression through the medium of the peripheral nerves alone. And, so far as the acute local lesions are concerned there should be no difficulty. But many chronic diseases do not present, especially in the first stage, symptoms of any urgency whatever; they are not accompanied by any constitutional disturbance and the insidious progress of the disease is very suggestive of the manner in which the lesion is undermining the tissues involved. In the acute local lesion, the vascular plays the important part, and attention is chiefly directed to it, both as regards symptoms and treatment. On the other hand, in some forms of chronic inflammation, and especially in certain lesions of the articulations, the neural element predominates and the vascular symptoms are, in many cases, not at all apparent. These facts being duly recognized, it becomes clear why so many of the so-called hysterical, surgical cases resemble an insidiously progressive chronic disease; for with chronic inflammation there is always a localized disturbance of function, associated with other symptoms which in many instances are very obscure and of uncertain etiology, while in the hysterical state there is also localized disturbance of function associated with manifestations which possess, in many instances, all the important characteristics of local inflammation.

For many years the effect of the emotions, not only in producing, but also in relieving abnormal states, has been recognized. In Burton's *Anatomy of Melancholy* may be found references to the value of this factor both in diagnosis and treatment. Many other works could be referred to, illustrating this point, with interest and profit. But this is scarcely the occasion for an extended research into the literature of this subject. Nor will it be of service to attempt an analysis of the relations of the three great nervous centers to the phenomena which are generally described as hysterical. We would rather say with Dr. Reynolds that "the essential fact of Hysteria is in the disturbed balance between the voluntary and involuntary power. Volition is defective; emotional, sensational and reflex activity are in excess, and the distortion may be brought about by the many divers circumstances of age, sex, position, employment,—but the precise nature of the change which is the efficient cause of such distortion, *i.e.*, the primary physical fact in the pathology of Hysteria, has yet to be discovered."[1]

Tuke remarks: "We have seen that the influence of the mind upon the body is no transient power; that in health it may exalt the sensory functions, or suspend them altogether; excite the nervous system so as to cause the various forms of convulsive action of the voluntary muscles, or depress it so as to render them powerless; may stimulate or paralyze the muscles of organic life, and the processes of nutrition and secretion;—causing even death.[2]"

It will not be difficult for us, therefore, to appreciate the effect of emotional activity upon the voluntary muscles. Indeed, all the sensations of which these muscles are capable, all the movements they may be made to undergo in response to the will, may be excited by this emotional cause, or voluntary muscular action may be apparently wholly lost through the same disturbing element. Still more, the various morbid sensations of the surface may be produced. Even superficial vascular changes may be brought about, inducing a local hyperæmia or an ischæmia. With defective volition, an excess of emotion, undue activity of sensation, and an easily excited reflex movement—especially if complicated by precocity in the child, or mental exhaustion in the adult, a long and intricate series of symptoms may follow, including disturbed muscular action, varying from the most extensive and general convulsion to the semi-tetanized muscles of one of the extremities, and from an apparent hemiplegia to the loss of power over a group of flexors. If these facts be appreciated, we can wholly coincide with Tuke when he says that "there is no sensation, whether general or special, excited by agents acting upon the body from without, which cannot be excited also from within by emotional states."[1] And to this statement we may add: There is no articular deformity depending upon chronic inflammation or disturbed muscular action—be it either loss of power or contraction—which cannot be simulated, and not unfrequently is simulated in the conditions which I have briefly attempted to describe.

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[1] *A System of Medicine*, edited by J. Russell Reynolds, M. D., vol. ii, p. 101.

[2] *Influence of the Mind upon the Body*, by Daniel Hack Tuke, M. D., M. R. C. P., p. 395.

The early writers on diseases of the joints fail to mention the existence of that variety of articular deformity which is associated with a more or less marked hysterical diathesis. Since the time, however, that Brodie called attention to the circumstance that many serious pathological conditions might be closely simulated by the condition which Skey[2] recognizes but does not name, except to call it Hysteria, which Paget[3] more recently describes as Neuromimesis, and which Esmarch[4] calls *Gelenkneurose*, the fact that these peculiar disturbances of the nervous system may successfully imitate the more serious deformities has been recognized; though, strange as it may seem, some of the more recent and pretentious works on the subject of joint disease and club-foot do not even refer to the fact that there are such conditions as hysterical deformities, while others dismiss the matter with only a passing notice.

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[1] *Op. cit.* page 146.

[2] *Lectures on Hysteria*, New York, 1867.

[3] *Clinical Lectures and Essays*, New York, 1875.

[4] *Ueber Gelenkneurosen*, Kiel, 1872.

In very many important respects the false so closely resembles the real disease that even the most experienced are, at times, at loss to decide whether, for instance, a given articulation is in a condition of progressive chronic disease, or simply in a neuromimetic state. Esmarch, after calling attention to the difficulties in diagnosis relates that for many weeks he was undecided regarding the state of an ankle-joint, which ultimately proved to be in a condition of *caries sicca*, and required amputation. Skey's testimony on this point is forcibly stated as follows: "It may be asserted with truth that every part of the human body supplied with nerves—be they cerebral, spinal or ganglionic—may become, under provocation, the seat of local symptoms so closely resembling the real disease to which that part of the body is liable, as to appear identical with it, and the resemblance to which is so perfect as to deceive the best of us." Paget says, after enumerating some of the hysterical conditions:—"And there is scarcely any of these disorders in which the mimicry of real disease is not sometimes so close as to make the diagnosis very difficult."

If these cases were rare they would be regarded as singular phenomena, but their frequency takes them from the list of even infrequent maladies and calls for recognition from all, since each one of us is apt to meet them any day. Brodie makes this remarkable assertion—which Esmarch fully endorses: "I do not hesitate to declare that among the higher classes of society at least four fifths of the female patients who are commonly supposed to labor under disease of the joints, labor under hysteria and nothing else." Skey, who obtained much of his knowledge of hysteria in St. Bartholomew's Hospital, "includes a large proportion of the lower classes," and says, "in reference to spinal affections in young persons I unhesitatingly assert that the real disease is not found in a greater proportion than one case in twenty—and even this is a liberal allotment."

My own experience convinces me that neuromimetic joints and spines, and more particularly the latter, are very frequent both in the upper and lower classes, and especially at that age when hysteria is most likely to develop. In other words, I may say that many cases of simulated disease of the articulations are not recognized as such, and it is a fair presumption that some, perhaps many, of the remarkable "cures" of which we hear now and then have been cases which, while presenting all or, at least, the more suggestive symptoms of the lesion, were simply in a state of neuromimesis. And this statement is notably true of the class of charlatans who profess to have, by inheritance or otherwise, the ability to cure these diseases by processes peculiar to themselves.

The cause of these erratic manifestations of the nervous system is very obscure. That it is not "mimicry" in the literal sense of the word is proven by the fact that the majority of my own cases have never had the opportunity to become acquainted with the symptoms of the disease itself. The causes assigned by the patients have been those which are ordinarily looked for in a history of chronic joint disease. A fall, a sprain, or over-exercise in skating or walking, in the majority of cases, forms the occasion for the first manifestation of the symptoms, though, as in the real disease, a history of traumatism is often wanting. After the symptoms once find a local expression, they partake of all the pertinacity and chronicity of joint diseases, and whether the muscular disturbances partake of the character of an intermittent *contraction*, or of the typical hysterical *contracture;* whether the general nervous symptoms are partly held in subjection by will power alone, or give evidence of functional disturbance by various erratic, emotional or typical hysterical symptoms, the solution of the enigma lies in a familiarity with the natural history and course of the real disease.

I have deemed it best in presenting a few of the cases I have met with, to follow the regional method, to bring out as fully as possible the symptoms, and to compare them with those of the chronic lesions of the joint involved. In following this plan I propose to give a full history of each case,—fuller, perhaps in some instances, than some might think necessary; but as many of the differential points are interwoven with the manner in which the history was developed, I have thought it proper to give a few of the histories practically unabridged. As the knee-joint affords an excellent opportunity to study both the real and simulated states, I will present some cases of neuromimesis of this articulation first. We will then consider the hip and spine, and lastly, club-foot,—giving comments, differential tables and conclusions in their proper places.

Case 1.—While on a visit to Troy, N. Y., in December, 1876, I was asked by my friend, Dr. W. P. Seymour, to see with him a little girl of 5 years of age, the daughter of a clergyman living near the city, who had been troubled with some very suggestive symptoms affecting the hip- and knee-joints on the left side—following a fall—and which had existed for several weeks. I was introduced to the patient at dinner and had the opportunity of watching her as she sat at the table. She was evidently a precocious child and mentally very active. She had that peculiar complexion which is supposed to be indicative of the strumous habit. I also noted that she had large eyes, with long eyelashes, bluish sclerotic, pale skin, showing the temporal veins very clearly, and a luxuriant growth of hair. These facts, with the characteristic gait and attitude of chronic joint disease, which were noted as she left the table, led me to infer that our patient had some serious chronic inflammation of either the hip or knee. Indeed, a diagnosis of morbus coxarius had already been made by a prominent surgeon of Albany, who had examined the case a few days prior to my visit; a diagnosis, however, which Dr. Seymour had rejected. After dinner a critical examination of the patient was made. In the standing position there was at once apparent a slight discoloration of the tissues over the knee-joint, a marked flexion of the articulation (about 35°), and a perceptible atrophy of the thigh and leg. The patient stood in the attitude characteristic of hip disease, with exaggerated flexion at the knee. Almost the entire weight of the body fell upon the unaffected limb, and there were evident the lowered gluteo femoral crease, the flattened natis and the tilting downward of the pelvis on the affected side, which makes the limb seem apparently longer, and produces the symptomatic lateral curvature of chronic coxitis. When asked to bend forward to pick up a key from the floor, the patient carried the entire limb, in its deformed position, backward with the pelvis, refusing to bend in the slightest degree either the hip or knee. When the patient walked, little or no motion was apparent at the hip-joint, the knee was neither flexed nor extended, and the limp was very marked and characteristic of chronic osteitis of the hip or knee. The patient was now placed in the supine position. It was found that the affected knee-joint was slightly warmer than its fellow, and that there was an almost complete obliteration of the fossæ on either side of the patella. There was tenderness on pressure over the condyles of the femur, and below, over the ligamentum patellæ. This tenderness was not present on the opposite side. The limb was then examined as to the passive mobility of the hip- and knee-joints. When the limb was moved as a whole, with the knee in its acquired position, no muscular rigidity existed at the hip. But if the attempt was made to flex the thigh on the pelvis, at the expense of motion at the knee, a very decided resistance was experienced at the hip. When the attempt was made to flex or extend the knee, it was found to be rigidly held in its deformed position by a very decided muscular contraction, and when a slight degree of force was used, the joint refused to yield, and the patient gave evidence of pain, both by facial expression and orally. The pain was referred to the inner side of the joint. A persistent attempt to flex the leg on the thigh, using a continuous rather than a great degree of force, was followed by a very perceptible, almost audible "click," which, Dr. Seymour remarked, reminded him of the sensation imparted to the hand as a knife blade passes its half opened position. Motion now, inside of the degree of flexion at which the limb had been habitually held, was free and unaccompanied by pain, but any attempt to extend the limb past its usual stopping point, was met by a decided muscular resistance, and apparently gave pain.

Persisting as before, and using a continuous force, gradually increasing it, and diverting the patient's mind in the meantime by telling her an amusing story, the leg passed the apparent obstruction, again accompanied by the "click." Now, again, we found free movement. These manipulations were repeated many times, and always with the same result. The leg would be arrested, either in flexion or extension, at exactly the same point, and the suggestive "click" would invariably occur as this point was passed. After these manipulations the patient was asked to move about the room. It was found that she could walk better than before. I may remark also that during the examination the patient showed considerable emotion, which did not, however, find expression in tears.

There were no hyperæsthetic spots over the spine. There was no history of masturbation, and the genital organs showed no evidences of irritation. The child had always been mentally over-active for her years, and there was nothing of importance developed in the hereditary history. Her general health had always been good, with the exception of the usual diseases of childhood, which she had always borne well. There was a history of disturbed sleep.

The previous treatment of the case included counter-irritation and almost perfect quiet of the affected limb. What proved to be over-anxiety on the part of the parents and friends of the patient, had directed a great deal of attention to the child, who in her emotional condition was, not unnaturally perhaps, not averse to this excess of attention.

Remarks.—The differential diagnosis involved a consideration of three conditions—the hip being excluded, as no positive symptoms of any lesion of this articulation presented. I. An osteitis or chondritis of the knee, following some injury to the cartilage at the time of the accident, and which would account for the "click." II. A chronic synovitis, not involving the bone, but attended with anomalous symptoms. III. A neuromimesis. After duly considering these conditions, the last was diagnosed, and, for the following reasons:

1st. A traumatism sufficient to lacerate the cartilage, and cause a mechanical impediment to motion, would have been attended with urgent, acute symptoms. 2dly. An osteitis dependent upon an injury or otherwise, would have been followed by more expressive symptoms, and the muscular rigidity would have been uniformly present, and would have prevented motion in the extremes of flexion and extension—not at a point between these extremes. 3dly. The swelling was subcutaneous, and, as was afterwards proven, due to the effect of the counter-irritation, thus eliminating the synovial membrane from implication. 4thly. The pain was demonstrated to be hyperæsthetic, and the heat was inferentially supposed to be a simple, local hyperæmia, due to the counter-irritation. As it disappeared in a few days, this supposition seemed correct. 5thly. The child was very emotional during examination, and would permit various movements of the limb when her attention was directed to other matters, which motions she would not allow when her mind was concentrated upon the joint. 6thly. The atrophy was no more than would result from the enforced disuse of the limb for several weeks. 7thly. The limp, attitude, etc., were the result of the position of the joint, which position, though fixed during the day, was modified during sleep, free movement of the articulation being then possible without the "click."

I have attempted to explain the peculiar "click," which forms the prominent peculiarity of the case, by supposing it to be due to the reduction of a temporarily displaced tendon, or perhaps to the reduction of a slight subluxation, in either event caused by muscular action. This latter condition I have seen in one other case.

The diagnosis was a surprise to the mother of the patient, for she had been led to infer, upon what we all would esteem good authority, that a serious chronic inflammation of the hip-joint existed. Dr. Seymour wholly coincided with me in my opinion. The parents were directed to stop all local treatment, excepting daily manipulation of the joint. The child was not to be asked any questions regarding her knee, and the excess of attention she had received was to be discontinued. The patient's thoughts were to be diverted from herself; plenty of exercise in the open air was insisted on, and a general tonic course of medication was advised. A few months later the mother of the patient called upon me, and stated that for a few weeks only slight improvement was noticed, but that afterward, under the stimulus of a promise that if the patient would walk "perfectly straight" by a certain time she might attend a Sunday-school festival in which she was greatly interested, the improvement became more rapid. She did walk without any limp; there was no pain, and the recovery was complete. There has been no return of the trouble since that date.

This patient exhibited symptoms which would suggest a primary local lesion of the knee-joint involving the vascular system, heat, swelling, redness of the cuticle, and pain, following an injury; adding to these, immobility of the articulation, limping, muscular atrophy and disturbed sleep, and all the important symptoms necessary to a diagnosis of chronic disease of the joint were present.

Case 2.—G. L. R., a boy of 12 years consulted me on November 25, 1878, introduced by letter from a prominent surgeon of western New York. The patient came into my office leaning on the arm of an attendant, limping very badly and complaining greatly of his left knee.

Inquiry developed no tendency to hereditary disease of the articulations, no history of phthisis or rheumatism, and there was nothing of importance in his early history, —he had always been a healthy, though not a rugged boy.

In February, 1878, the patient fell upon the ice in front of his home, striking upon his knee-joint. The injury was quite severe and the boy was carried into the house. There was great pain, followed by ecchymosis and some heat and swelling. These symptoms continued for a few days, and after their most acute phase subsided, there was more or less pain on moving the joint, with swelling at the point of injury. He remained in bed for five weeks, and for the two or three weeks following he was dressed, but hobbled around saving, in every possible way, the affected joint. Finally, about ten weeks after the injury he walked as well as ever, and during the spring and summer walked to and from school daily, a distance of four miles.

On October 10th, he went on an errand for his mother, and on his return his knee pained him very much and he was obliged to stop walking. After this, he "hopped around," always holding the leg flexed on the thigh in one position. From October 10th to November 25th (the day he consulted me), he had not been out of doors, except to come to the city. There had been no acute pain in the joint when it was at rest, but any sudden motion produced "sharp pain": he had slept tolerably well; there was considerable atrophy of the thigh and the leg muscles and the boy was thin and anæmic and, evidently, very apprehensive about his knee. The joint was held quite rigidly by muscular action in the extremes of flexion and extension, but examination proved that no real muscular spasm existed: the joint could be flexed and extended normally by persistently using a very moderate degree of force, though the attendant states that the position of the leg was not modified during sleep.

The patient gave a history of mental overwork: had studied hard and for several years: tastes had always been effeminate—always very gentle in his play, preferring the society of girls to that of boys of his own age; not emotional as to tears, but is what would be called "a nervous boy."

Remarks.—My comments on this case will consist simply of extracts from the letter which I sent to the family physician: "I may summarize the results of my examination as follows:—no marked pain on motion which was not limited by any reflex muscular spasm, though the spasm was closely imitated in the extremes of flexion and extension: no strictly involuntary symptoms at all, either nocturnal or diurnal: no swelling except, perhaps, a very slight one under the ligamentum patellæ, where the thickness of the cuticle (from iodine) made palpation very unsatisfactory: local temperature 2° lower than on opposite side: electrical reaction of muscles of leg and thigh normal, the same on either side: defective nutrition of limb due to disuse solely." . . .

"The boy is mentally very acute and physically very sluggish. I find he is inclined to avoid boyish occupations and consider his lack of stamina and pasty complexion to be not of the strumous sort. In some respects our patient is what may be called a typically neurotic boy."

"I would advise a gradual increase in the use of the joint and limb, for a few days passively using electricity, massage, etc., and then I would put the patient on his own resources entirely: he is to be asked no questions as to his feelings or sensations: give also, a thorough course of iron, cod-liver oil and malt, and keep the boy out of school and at out-door sports for at least one year."

The patient wholly recovered in a few weeks and has grown six inches since I saw him.

A diagnosis of chronic joint disease had been made in this case by a prominent surgeon, whose contributions to surgery have more than once been quoted abroad, and this gentleman was about to apply apparatus to the patient, rest and counter-irritation having resulted only in an increase of the symptoms.

Case 3.—Miss K., a young lady of good physique, residing in New Jersey, consulted me in February, 1878, in reference to what were apparently severe and urgent symptoms of disease of the knee-joint, the patient presenting herself on crutches.

She gave the following history:—About four years ago she began to have pains occasionally in the left knee: they were very acute, occurred at irregular intervals and were increased by walking. There was no limping at first. The pains came and went without any recognized cause and were as frequent at night as by day. She never awoke with pain and was, as a rule, free from it up to 9.30 o'clock, A.M., when it would generally occur: there was never any swelling of the joint which the patient could detect, though it was frequently hotter than the sound one: she was always able to move the joint with more or less freedom though at times with considerable pain: passive motion was accompanied by considerable resistance in the extremes of flexion and extension. After some months the symptoms became worse and the family physician ordered crutches which the patient has ever since used, without, however, the relief which was anticipated. Accompanying these local symptoms were other manifestations, affecting, especially, the head and neck. The sensations here were those of "trouble in the back of the neck"—pain, not increased by motion, a sensation of heat "like a hot plate," in the cervical region, but no headache. It sometimes happened that when the knee did not ache, the neck did, and *vice versa*. The pain in the knee was not superficial but was referred to the very "center of the joint." It was either a "gnawing or a lancinating pain":—there were no hyperæsthetic areas and, at the time of the examination, the local and general temperatures were normal. The patient did not look at all ill, and there were no facial traces of suffering so frequently seen in chronic osteitis of the joint ends: but there was hereditary history of phthisis, the mother having died therefrom. The case had been under the care of one who would be recognized as a competent observer, who, attributing too much weight, in the absence of specific symptoms, to the phthisical history, had made a diagnosis of incipient osteitis of the knee. There were symptoms which suggested this lesion, but like all purely neurotic symptoms, they were extremely irregular and inconstant, and this very element was the basis of an exclusive diagnosis in the case.

The crutches were dispensed with and the patient, who was an indefatigable teacher, was removed from her routine duties. It took the patient five days only to discontinue the crutches, though she had used them continuously for months and had not gone out of the house for the three months preceding Christmas, 1877:—this discontinuance did not produce pain and their absence, after the novelty wore off, was pleasant rather than otherwise. During the past summer the patient has been at the seaside and among the mountains, has used no artificial support whatever, and has returned greatly improved.

I may remark that this case is typical of that kind of "hysteria" which develops in those who are physically robust but who are subjected in their occupations to constant physical and mental strain. This lady filled an important position in a large school and gave almost incessant attention to her duties. I draw attention to this and, in noting the absence of any history of traumatic influence, to the difference that exists between the simulated joint lesions dependent on mental overwork and strictly emotional cases with the history of traumatism. In the first there are symptoms which find a local expression in other parts of the body, while in Cases 1 and 2, there are first local injuries through which the fear and other emotions of the patient seem to find an exit.

During the fall of 1875 my attention was especially directed by a case which occurred in my service at the Orthopædic Dispensary (Case 4) to the fact—which I had before noted—that the expressive muscular atrophy which occurs in morbus coxarius did not exist in the hysterical state which simulated it. In this particular case a slight atrophy only occurred even after several weeks of treatment which involved complete disuse of the limb and the pressure of adhesive plasters and bandaging, all of which are the necessary accompaniments of the mechanical treatment. This led me to investigate the electrical condition of the muscles in both the hysterical and diseased conditions of the hip-joint especially. The result was that in the former I found in all the cases I examined a normal degree of muscular contraction in response to the faradic current, while, in the latter, even in the earliest stages, and apparently coincidental with the first appearance of the reflex muscular spasm, there was a marked reduction. After many experiments, including the same tests as applied to voluntarily tetanized muscles, and which showed a normal reaction like the hysterical, and all of which uniformly led to the same conclusions, I invited my friend, Dr. E. C. Seguin, to apply the same test to several cases of hip-joint disease. This he did on May 17, 1877, and the result of my own observations, sustained by Dr. Seguin's experiment, are duly recorded in the *Archives of Clinical Surgery*, for June, 1877. From this article[1] I may be permitted to quote the following sentences: "The atrophy which occurs from simple functional inertia, combined with the pressure produced by adhesive plaster and bandaging, is witnessed when the not infrequent error is made of treating by these means and an apparatus, a neuromimesis of a joint lesion, for the real disease. This simple atrophy following disuse and pressure is altogether different from that which ensues from an actual joint lesion, and is unaccompanied by a loss of electro-muscular contractility."

These observations were based wholly upon original research, and while, so far as I know, I was the first to apply the electrical test as an aid in the differential diagnosis of true and false joint diseases, it remains for me, as a matter of justice to Charcot and Esmarch, as well as to myself, to state that Charcot mentions that in a case of hysterical contracture "the electrical contractility of the muscles has remained nearly normal," and Esmarch, in his essay on "*Gelenkneurosen*" remarks, "Notwithstanding, the muscles maintain a well nourished condition, and retain after long disuse, their electrical reaction." At the same time I was studying this subject, Dr. Émile Valtat of Paris, was engaged in experimentally demonstrating the muscular atrophy of joint disease[2] and reached in some respects the same conclusions as my own, as applied to the condition of actual disease. After these statements it becomes unnecessary for me to call attention to the value of the faradic current, as a means of precision, in the diagnosis of the neuromimetic condition.

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[1] "Reflex Muscular Contraction and Atrophy in Joint Disease, etc.", by Newton M. Shaffer, M. D.

[2] "De l'Atrophie musculaire consécutive aux maladies des articulations," Paris, 1877.

In Seguin's "Series of American Clinical Lectures,"[1] I have given parallel tables showing the differential points in diagnosis of typical cases of chronic synovitis and chronic osteitis of the knee-joint. I will reproduce them here, make one or two additions to them, and follow them with remarks on the diagnosis of the neuromimetic state, as applied to the same articulation.

CHRONIC SYNOVITIS OF THE KNEE-JOINT.[2]	CHRONIC ARTICULAR OSTEITIS OF THE KNEE-JOINT.[2]
1. Capsule thickened,—Effusion marked.	1 No thickening of capsule evident,—No sense of fluctuation.
2. Natural contour of leg and thigh,—Joint outline obliterated.	2. Muscular atrophy marked,—Joint outline clear and distinct,—Joint appears large, on account of the diminished size of both thigh and leg.
3. Motion extensive and nearly normal.	3. Motion *nil.*
4. Resistance to motion *elastic,* and efforts to overcome it not productive of pain.	4. Joint held perfectly rigid by muscular action alone.
5. No reflex muscular spasm present.	5. Reflex muscular spasm affecting both flexors and extensors.

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[1] See author's comments (Sept., 1923) on page 181.

[2] *The Etiology and Pathology of Chronic Joint Disease.* Vol. iii, No. vi.

6. No pain present, nor produced by forcible tests.	6. Acute pain upon the slightest attempts at joint motion.
7. No perceptible limp or hesitation in walking.	7. Unable to walk from pain and deformity.
8. Sleep normal,—No reflex osteitic cry.	8. Incoherent cries and "starting pains," occurring during sleep.
9. Femur and tibia in normal relation to each other.	9. Tibia subluxated backward (partial) by muscular action.
10. Symptoms local, so far as those *dependent* on the joint lesion are concerned.	10. General and local neural symptoms directly referable to the joint lesion.
11. The superficial tissues over the joint may be either slightly warmer, or the same as the healthy joint.	11. Local temperature rarely lower than normal—almost always increased from ½° to 3°.
12. Electrical contractility of leg and thigh muscles not impaired before the occurrence of the reflex muscular spasm.	12. Electrical contractility of leg and thigh, muscles reduced;—in many cases remarkably so, even in the early stage.
	13. The reflex muscular spasm is not modified during sleep—nor does it yield to the ordinary doses of opium or chloral.—It will yield, however, wholly to the profound anæsthesia of ether or chloroform.

We will now consider the various points in this table as applied to the hysterical condition of the same articulation; first, however, calling attention to the fact that in certain conditions a hysterical *contracture* may exist, especially in adults, which more closely resembles the typical reflex spasm of chronic osteitis, than the emotional *contraction*, which I have attempted to describe. This *contracture* will be specially considered later.

1. Thickening over the joint may or may not be present; when present, it is very evidently confined to the cellular tissue only, and is generally accompanied by hyperæmia and hyperæsthetic areas, which latter are not, as a rule, confined to the immediate region of the joint, or to points where tenderness should be developed by pressure either in synovitis or osteitis.

2. Atrophy from disuse only; not the expressive wasting of chronic osteitis.

3, 4 and 5. The joint may possess even an abnormal degree of mobility, as in hysterical paralysis, but it will generally be found that motion is impeded at first by muscular action, which may either closely resemble the tetanoid spasm of osteitis, holding the joint absolutely rigid at a fixed point, as in Case 1, or it may permit joint movements, which are arrested just inside of the extremes of flexion or extension, as in Cases 2 and 3. In any event persistent effort, in the meantime diverting the patient's attention, will wholly overcome the muscular resistance. If the patient be examined on two or three occasions, the muscular conditions may be found variable, and certain movements which the patient will almost invariably resist under passive examination, will be voluntarily executed when the patient imagines himself unobserved.

6. Pain may be apparently very severe, and at points where it usually exists in osteitis. Generally, however, this pain is just as severe when the subcutaneous tissues are slightly pinched, as when firm pressure is made. In some instances pain can be developed at any point by simply directing the patient's attention to it. The oral expression of pain is wholly different in the hysterical condition from

that which occurs in osteitis. In the former it partakes more of the *voluntary* character; the latter is *involuntary*, and is accompanied by a facial expression which cannot be counterfeited.

7. There is always a limp if the limb is used at all. In many cases the fears of the patient, reinforced by the apprehensions of the physician, place the patient on crutches or in a splint. The limp may closely resemble that of osteitis, or it may partake of the character imparted by congenital dislocation of the hip. If examined with care, however, there will always be found a sort of exaggeration about the gait, and in some cases the patient, under some stimulus, will involuntarily resume command of the hysterical muscles, and walk well for a few moments.

8. Sleep may be disturbed. In the majority of cases, it is normal. An incoherent cry, resembling that of osteitis, occurred at night in one of my cases, but it was evidently due to intestinal irritation. The "starting pains" do not occur in neuromimesis. *Consciousness* is necessary to form the group of symptoms which we are studying now.

9. The position of the limb is, as a rule, flexed—though I have seen it held in extreme extension—a position which Esmarch says is very frequent. At night, during sleep, the malposition may be, almost always, readily overcome.

10. In almost every case the patient will be found to be emotional to a greater or less degree, and there will exist some general or specific evidence of this condition in the history, conduct or appearance of the patient.

11. If the affected joint be hotter, a superficial hyperæmia exists. In Case 1 it did exist, and was apparently due to the counter-irritants employed; and yet in Case 2, where counter-irritants had been thoroughly used, the local temperature was 2° lower. In two other knee cases, not

reported, there was a reduction of 2° in one and 1½° in the other. The hyperæmia of neuromimesis, is apt to disappear and again reappear, while the temperature of an inflamed joint varies but little from day to day.

12. In the hysterical joint, the muscles retain their normal contractility, as tested with the faradic current. Even if the hysteric contraction has existed for a long time, the faradic current shows little or no reduction either in the muscle or nerve currents.

13. A full dose of opium or chloral will cause the muscular contraction to yield, should it apparently resist in natural sleep.

From these facts it will be seen that all the important symptoms of disease of the knee-joint may occur in a neuromimesis. But from a comparison of these differential statements we may draw the following conclusions regarding both the true and false knee-joint conditions:

I. The neuromimetic condition resembles both the chronic inflammation of the synovial membrane and the bone. If it be remembered, however, that the changes which take place in chronic synovitis produce very few, if any, subjective symptoms, while the objective are prominent, as applied to the joint itself; and that the hysterical imitation presents a long train of both subjective and objective symptoms and signs, with the former in excess, but little difficulty will be experienced in making a differential diagnosis.

II. If the following conditions exist, a diagnosis of chronic osteitis of the knee-joint may be made with certainty:

1st. A muscular spasm, which cannot be overcome by persistent effort, while the patient's mind is diverted—a spasm that does not vary night or day, whether affecting motion in the extremes of flexion and extension only, or simulating an actual synostosis—a spasm which is not affected by the ordinary doses of opium or chloral, and which yields to profound anæsthesia only. 2dly. A markedly reduced faradic reaction of the muscles thus affected. 3dly. A localized and uniform rise of temperature over the affected articulation. 4thly. The presence of purely *involuntary* neural symptoms, as shown above, in the reflex muscular spasm, the starting pain, the osteitic cry, etc.

III. If the following conditions are found, a diagnosis of hysterical knee-joint disease may be made with equal certainty: 1st. A variable muscular rigidity or contraction which can be overcome by mildly persistent effort, while the patient's mind is diverted, or which yields during natural sleep, or which wholly disappears under the usual doses of opium or chloral. 2dly. A normal reaction of these contracted muscles in response to the faradic current. 3dly. The absence of a rise of temperature, or especially the presence of a reduced temperature over the affected joint. 4thly. The presence of various emotional and semi-voluntary manifestations, which are variable and inconstant—the variability and inconstancy being due to the different conditions of the emotions, as affected principally by volition.

We will now consider neuromimesis of hip-joint disease.

Case 4.—Fanny A. is 15 years old and lives in New York.

Hereditary history.—Father an epileptic; is an inmate of the Blackwell's Island Hospital for Epileptics, and "is fast becoming an idiot." Mother is healthy; the patient has one brother who is well; no sisters.

Patient is a plump girl; has a clear skin, and is not anæmic; is an inmate of a Home for Friendless Girls, and applied for relief at the Orthopædic Dispensary Aug.

3, 1875, accompanied by the matron of the Home. Six months previously, while in domestic service which required much running up and down stairs, she was attacked by a severe pain in the knee of the right leg, attended by a very perceptible limp. The pain soon involved the hip-joint, and finally the back. The pain was frequently urgent at night, and "the leg was drawn up and in" both night and day. There was no history of any direct injury to the affected limb or joint.

The patient applied at the dispensary during my summer vacation, and the following notes were made in the record book by the assistant surgeon who received the case:

"Condition of limb, adducted and slightly flexed; muscular rigidity and pain at thigh and knee; flattening of natis and alteration of gluteo-femoral crease. Pressure through trochanters gives pain; motion in any direction, especially adduction and abduction is resisted and gives pain. Pain on going up and down stairs; pain at night; limps badly; slight concussion to heel causes apparently severe pain. Menstruation regular and normal. General condition of patient seems excellent; she came into the room bearing almost her entire weight upon the arm of her attendant."

A diagnosis of hip disease was made by the assistant surgeon, and a note to that effect was sent to the managers of the "Home."

On August 16, 1876, the hip splint was applied. On the 20th the following record was made in the history book:

"Relieved of pain; walks very nicely with the splint."

August 28th.—"Muscular rigidity still well marked."

September 11th.—"Doing well."

September 15th.—"Examined to-day very carefully by the surgeon in charge, and hysterical symptoms established beyond doubt. Splint and appurtenances removed and patient ordered to walk home."

"The following conditions were present during the examination: Patient, under the instructions of the operator, voluntarily flexed, extended, abducted and adducted the limb normally. She resisted at first, but gradually yielded and permitted the operator to place the limb in the extreme positions mentioned above, including also all degrees of rotation and circumduction. Pain only on pressure over anterior superior spine and above crest of ilium. The patient walked away without apparatus, and with slight limp." No special treatment was adopted. She was ordered to use passive movements; a tonic was prescribed, and the case was kept under observation. On October 1st of the same year she went to Hastings-on-the-Hudson to fill the position of a domestic, wholly cured, and there was no return of the trouble even after the hard work incidental to her duties. She called at the dispensary during the past summer (1879) to consult me regarding a pain in the back, which proved to be a localized hyperæsthesia—a condition which did not exist during the previous treatment of her case. She told me that her limbs were equally strong, and that the ailing hip had given her no pain or uneasiness for many months.

Remarks.—The symptoms in this case were well calculated to deceive, and had the patient been "coached" by an expert they could scarcely have been more closely simulated; and yet this girl had never seen a case of hip-joint disease, and knew no more of its symptoms than does a child of three years. If further confirmation was needed to sustain the correctness of the diagnosis, note the effect of the extension apparatus: "Relieved of pain, and walks very nicely with splint,"—its almost invariable effect in true, chronic coxitis. There was no disturbance of uterine function, no symptoms of spinal irritation, no hysterical convulsions, no particular emotional disturbance; but there was an indescribable something about the patient as she walked into the dispensary with the apparatus applied—as I first saw her—which suggested neuromimesis. There was the absence of the relation of cause and effect regarding her attitude and other expressions of her condition: a voluntary, and therefore, irregular, effort to accommodate the one to the other, characteristic of the false as distinguished from the real disease, and the result of the examination fully confirmed my suspicion. The differential diagnosis turned principally on two points, viz.: the absence of the expressive atrophy of the thigh muscles and the absence of the *persistent character* of the reflex muscular spasm, which is an invariable symptom of chronic osteitis of the hip-joint. It was in this case that I first tested the thigh muscles with the faradic current, and found, as I have previously stated, a normal contractility.

It is worth while to call attention to the fact that in some cases of neuromimesis, as in this, the patient will express himself as greatly, perhaps almost wholly relieved after apparatus is applied; others wear their pads and straps so very tight that excoriations result, and insist that the appliances fail to give them comfort unless so worn—ignoring excoriations and other inconveniences which, in the true disease, often cause great uneasiness.

In other cases the apparatus is soon discarded, having produced no other effect than the trouble incident to its use, leaving the patient, apparently, much worse than when it was put on. I draw no distinction as to which class recovers the more quickly; the one just detailed made an exceptionally rapid recovery. I have now a case under observation, which was referred to by my friend, Dr. T. G. Thomas, where everything which could be suggested by the most emiment medical talent in the city was carried out in the treatment of a recognized hysterical paralysis of the right lower extremity, with only partial success. With apparatus the patient is now comfortable, and walks much better than before its use; but the question of complete recovery is one which the future only can decide.

Case 5.—Miss E. O., aged 20, residence, Greenpoint, New York, applied for treatment at Orthopædic Dispensary, September 29, 1875.

The patient's parents are living and in good health; has two brothers and two sisters, all in good health, except one sister younger than herself, who has suffered from chronic, suppurative hip disease for eight years, and who has been under the immediate care of the dispensary for several years. There is, apparently, a good family history with this exception, and no disease of the nervous system is known in previous generations.

The patient has always been "a delicate girl." Menses first appeared at 14—very irregularly at first, but recently a great improvement has taken place in this respect. Is in apparently good physical condition, though somewhat pale.

Last winter (1874), she fell while stepping from a street car, and "hurt her hip," beside "cutting her elbow and knee." The patient walked home, however, with a decided limp. Soon after she "wrenched her hip" badly while rolling a barrel of flour. This last accident proved more serious than the former, the patient being unable to walk after the second injury to the joint. She was carried into the house and placed in bed, from which she did not move for three days. During this time she suffered much pain in and about the hip, and after again assuming the upright position she walked with "a bad limp" and required assistance in going up and down stairs for several weeks. In the following spring (March) "pain in the back" appeared as an urgent symptom, the pain being located over sacro-iliac junction. The lameness and the pain were treated at home without success. In April she was seized with what the family physician called "neuralgia of the spine," and soon after hysterical convulsions appeared. Consultations were held, and "heart disease" and "tumor in the groin" were diagnosed. In this condition she went to Connecticut to visit a friend. While there she again "sprained her hip." This third injury to the hip-joint was followed by an increase of the limping, pain in the back, hip and knee, and very restless nights. She returned to New York on September 25th, the symptoms being much aggravated by the journey. The pain in the knee became worse, sleep was very irregular and greatly disturbed, "the limb began to draw up," and the knee rested against the opposite thigh. Crutches were obtained, and with their use and the assistance of her mother the patient presented herself for examination.

I was about leaving the dispensary when the patient appeared. Having an engagement, I did not stop to make a careful examination, but taking into consideration her marked expressions of pain, both facial and oral, as I moved the limb while the patient stood before me upon crutches, her attitude, the rapidly repeated history of traumatism, and the apparent extreme disability and muscular rigidity of the hip-joint and especially remembering that the patient's sister was under my care for morbus coxarius, I directed the house surgeon, Dr. George B. Packard, to apply a weight and pulley if manual traction should afford relief. The examination of the house surgeon is thus recorded:

"Patient pale and with anxious countenance; limps very badly; a great deal of hyperæsthesia of back, especially in lumbar region; vertebral column very flexible; no prominence visible; great tenderness in inguinal region; excruciating pain and apparent reflex contraction on movement of joint; flexion and adduction of thigh; flattening of natis; gluteo-femoral crease lower and larger. *Traction relieves pain.* Patient entered ward, and weight and pulley applied."

October 1st.—"Relieved a little, but does not sleep well. Ordered morph. sulph. gr. ¼ at night. Does not get much relief from pulley; complains of pain in each hip. The symptoms are those of hysterical hip disease, and a diagnosis to this effect was made by the surgeon-in-charge."

October 8th.—"Ordered a few drops of tinct. of cinchona bark as a substitute for the ¼ gr. dose of morphine. The effect was the same as though morphine had been given. The condition is unmistakably a hysterical one, and patient was ordered to get out of bed and to attempt to walk. She walked with great difficulty, using a chair in place of crutches. The hip symptoms have subsided entirely, and are now centered in the knee-joint, which has been held in extreme extension by the weight and pulley."

October 20th.—"Discharged in the following condition: Hip symptoms wholly removed, but the knee is held firmly fixed in the position it may happen to assume after manipulation. Considerable pain in the knee, and patient walking very badly."

December 2, 1876.—"The mother called to-day to report, and says she 'is sure the doctors dislocated the knee-joint while the patient was in the ward.' This 'dislocation' was reduced in the following manner: 'While rubbing the joint with a liniment, something suddenly snapped, and the following morning the patient could walk as well as ever before.'"

April 10, 1877.—"The patient called at the dispensary to-day with her sister's hip splint, which needed repairs. She walks perfectly well. Sleeps well 'with valerian once in a while.' Has pain in hip and knee during easterly storms and after very long walks. No difference in size or length of limbs."

Remarks.—All the symptoms in this case were very urgent, and I have never seen a closer imitation of the real disease. These symptoms were developed in a tolerably well-nourished girl, who had been the personal attendant of her afflicted sister for several years. This sister had passed through all the stages of a chronic, suppurative coxitis. The mimicry in this case is undoubted. The dread of the disease, of which she daily saw a very painful example, was sufficient, in her extremely sensitive condition, to develop the train of symptoms described, and which, at my first and superficial examination, deceived me. But when I again examined the patient, after the weight and pulley had been applied for two days, I found much the same condition of affairs as has been described in Case 4. When the first attempt to flex the thigh was made, the patient being in the supine position, an exaggerated expression of pain followed that would be difficult to describe, and the pelvis moved with the thigh as though an actual anchylosis existed at the hip-joint. Next I held the pelvis firmly, and strove to overcome the flexion. The pelvis tilted forward, and the tubera ischii, resting on the mattress underneath, became the fulcra by which the lumbosorsal spine was alternately lordosed or kyphosed. Abduction of the thigh was next tested. But the pelvis moved with the femur upon the opposite acetabulum at the expense of the vertebral column. But I became convinced from various over-expressions of pain and exaggerated statements as to the effect of some simple tests, coupled with the history of hysterical convulsions, that a neuromimesis of hip disease was present. While the patient was looking in another direction, I made a persistent, but not forcible flexion, and after a few seconds of determined resistance the joint suddenly yielded. So it proved regarding the other movements, until, after a time, free passive motion of the articulation was permitted without pain.

The patient was in bed. Buck's extension was applied to the limb, and the question arose: What shall be done? It was deemed best to continue the traction for a few days. This was done, and the patient then was *permitted* to get up. As mentioned in the notes, all the hip symptoms disappeared under this treatment, and the trouble concentrated about the knee-joint; and this complication did not yield prior to the discharge of the patient. The manner in which recovery was effected is very suggestive of the methods employed by the professional "bone-setters."

Case 6.—Charlotte K., aged 12: admitted to St. Luke's Hospital, May 13, 1879.

A portion of my own notes of this case have been mislaid and I am indebted to my friend, Dr. Robert Abbe for the following memoranda made by him when the patient entered the hospital.

Patient enters hospital in excellent general condition but complaining of much pain about the right hip on walking or sitting down. She has a marked limp and drops on that side when walking; she gives a history of having been injured on that side eight years ago, with pains and limping ever since. On examination there is:

1. Shortening three eighths of an inch by measurement of limbs.
2. Atrophy of solid tissues, the right thigh and calf being one fourth inch less in circumference than the left.
3. The natal fold on the right side is higher and deeper —the buttock slightly flattened.
4. The inguinal fold is drawn up on the right side and the genital fissure considerably drawn to the affected side.
5. Pain about the inguinal region and iliac fossa; likewise pain in pinching up the skin anywhere on either thigh or leg.
6. Some adduction and a little flexion of thigh on abdomen.
7. The patient refuses to flex thigh on abdomen when lying down, or to allow much flexion of it.
8. There is rigidity of all the muscles of the affected thigh.

Points on which an exclusive diagnosis was made.

1. The emotional element in the child; easily affected to tears, without pain. She seemed also very conscious of observation and suspicious of it.

2. The rigidity of muscles of thigh is variable, as when attention is directed thereto or diverted therefrom, etc.
3. The flexion of thigh which occurs when patient sits in a chair cannot be obtained when she lies down.
4. Patient can put on her own shoes and stockings.
5. She attributes disease to and dates back her trouble from an injury eight years ago.
6. There is more or less hyperæsthetic pain at most any point of opposite leg, or any part of body when pinched or touched.
7. Uniform temperature; no evidence of suppuration; good appetite; good general condition.
8. Psoas muscle was not involved in the contraction.

In a letter recently received from Dr. Abbe, he also says:

> "The child, when not conscious of observation was occasionally seen when playing with other children, to jump down from buttress of the front steps of the hospital—a distance of three feet, and to run off without evidence of pain. She was also, on one occasion, taken off her guard by an offer of money with which to buy peanuts from a passing vendor and momentarily developed an unexpected freedom of action about the hip-joint."

This case came into my service at the hospital with a diagnosis of hip-joint disease, and Dr. Abbe has described symptoms upon which such a diagnosis might easily be made, but the condition of the patient and the elements of the exclusive diagnosis are so plainly stated in the notes that extended comment seems unnecessary. In this as in the preceding case, the extremely variable character of the muscular contraction was the turning point in diagnosis; a wide difference existed between the symptoms developed at the formal examination and those shown by the patient when she thought herself unobserved.

I may mention one other point which would be of value were the elements of diagnosis more confusing; *i.e.*, the atrophy. It was the same at both leg and thigh. In true joint disease of several years' standing, the atrophy of the thigh is far in excess of that of the leg. I have made and recorded several hundred observations on this point and find such to be uniformly the case. The shortening of three eighths of an inch was undoubtedly congenital. This patient recovered under the treatment pursued in the other cases reported, and left the hospital in a few weeks.

Case 7.—D. P., æt. 10. Residence, New York State.

This patient applied to me on September 10, 1876, suffering from all the important and many of the urgent symptoms of hip disease—left side. The hereditary history, as related by her mother, was not very satisfactory, as nothing definite could be learned, other than the fact that the child's father was dead, and that "all the rest of the family were healthy."

The symptoms from which the patient suffered had been of insidious growth. She first commenced to limp, shortly after an injury to the hip. The limp had been followed by pain, though the mother states that the pain followed so soon, as to be almost coincidental with the limping. The pain was in the thigh, knee and hip, the patient passed through some sleepless nights, and gradually the limb had become "very weak and deformed." The limp and pain became so much worse that crutches were used. The patient used these crutches as she came into my office—with a diagnosis of hip disease, and a letter of introduction from a prominent surgeon of one of the Hudson River counties.

It was with great difficulty that the patient was persuaded to lie down upon a lounge for examination. After many efforts and a great many suggestions from the mother, the patient was finally placed in the supine position, the mother, in the meantime, making what seemed to be manual traction with a degree of force that indicated long practice. The patient, all this time, was shrieking with pain, and grasping the furniture near at hand, apparently as a means of counter-traction. I imagined that the case was one of chronic osteitis of the hip-joint, in the stage of exacerbation. After much persuasion I, at last, induced the mother to permit me to make the traction and control the limb. I then commenced to gently test the condition of the joint, as regards motion. While manipulating in the mildest manner, I was startled by an urgent cry from the patient, and an imperative command, "hold it tighter," two or three times repeated. I was already making all the traction possible, and naturally asked an explanation. The mother hurriedly said "you don't squeeze the ankle tightly enough." This threw a new light on the symptoms. Desisting wholly from all efforts at traction, I merely compressed the ankle-joint with all my power. While doing this I could place the thigh in any position, and could even press the articular surfaces together without resistance or complaint.

Still "squeezing the ankle," I was able to get the patient in the upright position with little or no trouble. Without any support but that afforded by her crutches, the thigh became flexed and adducted. The whole limb was visibly, though not markedly atrophied. There were various hyperæsthetic areas on the affected limb—principally on the inner aspect of the thigh—and over the crest of the ilium. Pain was produced by pressure through the trochanters, by crowding the sacro-iliac surfaces together, and by digital compression in the inguinal region. The patient stated absolutely that she could not walk without support. There was normal faradic contractility of the leg and thigh muscles.

I informed the mother that her child did not have hip disease—and advised the same course of treatment that had been successfully pursued in other cases. My diagnosis was not well received. The mother openly declared that she preferred Dr.——'s opinion to my own.

I had almost forgotten about the case when, one day, in passing through my ward at St. Luke's, I again met my former patient, still upon crutches, and still suffering from "hip disease,"—under which diagnosis she entered the hospital. It seems that the mother of the patient had wholly disregarded my advice, and had consulted a prominent surgeon of this city, who, after a careful examination, had diagnosed coxitis, and had said I "had made a great error." The especial attention of the house staff was called to the patient and my friend, Dr. G. A. Spalding made the following notes of the case, which he has kindly given me.

1. Patient entered hospital on October 30, 1876, using crutches, and refusing to stand upon, or use her left limb, in any way.
2. Thigh flexed and adducted. The attitude and position of limb were characteristic of hip disease.
3. Very marked expression of pain on passive motion of joint—principally at the knee.
4. Joint motion limited in every direction at first. It became almost normal when persistent effort was made while patient's attention was diverted. Apparent atrophy of thigh and leg muscles.
5. Patient very restless at night. She had used morphine prior to entrance to hospital.
6. When patient was under observation she complained much more than when she imagined herself unobserved.
7. *Traction relieved pain*, and apparently rendered patient more comfortable.

The peculiarity manifested when I first examined the limb, viz.: the preference for a squeezing sensation at the ankle had become greatly modified. The patient was now relieved by direct traction only.

The treatment pursued consisted in the removal of the crutches, passive movements, with cod liver oil and ferruginous tonics. She improved very rapidly and on December 20, 1876, she left the hospital without any evidences of hip disease. The subsequent career of this patient is thus described by Dr. Spalding in a letter to me:

> "On Feb. 19, 1877, about two months after her discharge, her mother again presented the patient for admission to the hospital, giving the following history: A few weeks previous she had been seized with convulsions. These convulsions, the mother stated, were becoming more and more frequent and alarming—as many as three or four occurring in twenty-four hours. As the mother gave this history, she was occupied in unrolling a large bundle, which proved to be a blanket. This she spread carefully upon the floor, remarking that the hour for one of these attacks had arrived, and that it was her custom to put the blanket down as a protective. Precisely at 11 o'clock A.M. the patient composed herself comfortably upon the blanket, and passed into one of the most characteristic hysterical convulsions I have ever witnessed. The subsequent history is very brief and most satisfactory. The usual remedies lessened the frequency and shortened the duration of these attacks. But an absolute cure was not affected until later. I chanced to be in the ward one day at the time the patient was seized with a convulsion, and happening to see a siphon of carbonic acid water, I picked it up and holding the young girl firmly by the back hair I discharged the contents of the siphon down her throat. Her convulsive movements were instantly checked, and she promised to avoid all such conduct in the future. She kept her word, and in a few weeks was discharged from the hospital. During all this time the patient had no recurrence of the hip-joint manifestations."

Remarks.—The hysterical diathesis was well demonstrated in this case, as Dr. Spalding's experience proves; and my own experience shows, as I have remarked in other cases, that a diagnosis of "hysteria," be it qualified by ever so many Greek names, is very apt to produce a not very pleasant impression, so far as the patient and her friends are concerned.

We observe also in this case the change in the symptoms, as relating to traction of the joint. When I first examined the patient, I explained to the mother, in presence of the child, that in true joint disease traction afforded relief, and that simply "squeezing of the limb" would not produce any effect upon true joint symptoms. When the patient entered St. Luke's she was fully prepared upon this point, and I have no doubt that this information assisted in deceiving the next examiner.

We are assisted in our study of knee-joint lesions by many favoring circumstances. We can detect by inspection alone any considerable change in the outline of the articulation. We can discover, oftentimes without the thermometer, a rise of temperature, due to intra-articular changes. Palpation is of great service, and the appearance of the superficial tissues is frequently of material assistance. But in considering the differential diagnosis of hip-joint lesions in the first stage, we cannot rely with the same certainty upon any one of these diagnostic aids. Inspection shows us a flattened natis, a deformed position, an altered gluteo-femoral crease, etc., all of which may arise from a variety of causes. The surface thermometer is rarely of service. The joint is too remote from the surface to make palpation available, in the first stage of the disease, in the great majority of cases, and the appearance of the superficial tissues is not of special diagnostic value. Our means of objective diagnosis are therefore limited at the hip, and we are obliged to depend upon a closer analysis of the symptoms, especially the subjective.

Bearing these facts in mind, and recalling the essential points developed in our study of the true and false lesions of the knee, especially as applied to chronic osteitis and neuromimesis, we may summarize our observations upon these same points as applied to the hip as follows:

In chronic osteitis of the hip[1] there exists, among others, the following symptoms.

1. *A limp*, which forms in the large percentage of cases the first symptom noticed,—a limp which is not expressive of fatigue,—which is more apparent after rest than after exercise, and which usually increases until the patient is unable to bear any weight upon the affected joint.

2. *Pain.* This symptom rarely antedates the limp, but may appear simultaneously. Generally it follows the limp after many weeks, sometimes months. The pain is usually referred to the knee, and is very frequently described as presenting at some point remote from the hip.

3. *A state of apprehension* regarding joint movements, difficult to describe. This apprehensive state always attends the pain, and frequently antedates its oral expression. It is almost invariably developed by passive motion if pushed beyond the point of muscular resistance, and is plainly demonstrated by some particular form of forcible movement—by concussion in some, by quickly rotating the thigh in others, etc.

4. *Muscular spasm.* This exists always as a symptom of chronic osteitis of the hip-joint, though its existence might easily be overlooked in the early stage of the disease by a careless observer. In this early stage, the one most likely to be confounded with neuromimesis, this spasm of the muscle is perceptible in the extremes of flexion and extension, and it is especially noticeable when, with the patient in the prone position and the pelvis firmly held,

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[1] Chronic synovitis of the hip-joint, is, probably, of rare occurrence and is not easily diagnosed. See the author's Clinical Lecture on the "Etiology and Pathology of Chronic Joint Disease," pp. 18 and 34, and remarks in "Pott's Disease, Its Pathology and Mechanical Treatment," by the author, p. 25 et seq. See also remarks by V. P. Gibney, M. D., in Article, "Dislocation of the Hip in Children."—*Amer. Jour. Med. Sciences*, Oct., 1879.

the flexed leg is used as the long arm of a lever to make rotation of the thigh *outward*. This muscular spasm increases, as a rule, with the limp, but many months, or even years, may pass before it reaches the point where all movement of the joint is arrested; and cases may occur where the spasm simulates actual anchylosis, and yet there may be a very slight limp only, and no oral expression of pain.

5. *A progressive muscular atrophy* of the muscles thus affected as shown, as has been already mentioned, by comparative measurements and the electrical test.

It will not be necessary to mention further points. They have been fully considered and discussed in our consideration of the knee-joint conditions.

In the neuromimesis of hip disease these symptoms present the following phases:

1. The limp is variable and suggests fatigue. The expressive conservative element so apparent in hip disease is lacking. This variable, tired limping is, as a rule, much better after rest, and is often absent in the morning after sleep. It almost invariably follows the pain, though in Case 7 it did not.

2. Pain is usually the first symptom, and it is found most generally in the immediate region of the joint—for example, over the iliac crest, or anterior superior spine. The hyperæsthetic character of this pain is easily demonstrated as a rule.

3. In place of an apprehensive state, in response to the tests applied, will be found a series of symptoms which are erratic and inconstant.

4. A condition of muscular rigidity often exists, but, unlike the true muscular spasm, it can, in most cases, be overcome in the manner before stated.

5. A very perceptible degree of atrophy may exist, such

however, as would, arise from inertia only. A normal electrical contractility exists in all the muscles of the thigh.

The conclusions reached in our study of the knee-joint lesions apply with equal force to the hip-joint, local temperature alone excepted.

Cases sometimes occur where actual hip-joint symptoms exist, associated with undoubted hysterical manifestations. If the former are not urgent, and the latter very evident, the difficulty of making a correct diagnosis is very great. The danger lies in ignoring the obscure, but real, and attaching a too great importance to the false. Such a case occurred in my own experience, and may be briefly related.

Case 8.—Miss A. S., a young lady residing in Brooklyn, of healthy parentage and with a good early history, passed through the ordinary symptoms of the first stage of hip disease, and came under my care in 1875. After a thorough mechanical treatment, covering about one year, all the symptoms of the joint lesion subsided, and in consultation with the family physician, Dr. A. W. Catlin, it was decided to remove the splint. This was done, and the patient walked without any limp, except that which would be expected from the disuse and confinement of the limb.

Soon after the removal of the splint, however, various typical hysterical symptoms manifested themselves, and hyperæsthetic areas developed in various portions of the affected limb, and especially over the crest of the ilium, the lumbar spine, the sacro-iliac synchondrosis, and the outer portion of the thigh. There were other symptoms, also hysterical, which developed at the knee-joint, and the emotional condition of the patient became very apparent. All of these pointed to a neuromimetic state. While in this condition, which existed for some weeks, the patient began to limp slightly, but the joint motions were, as before, nearly normal. The involuntary symptoms, especially the nocturnal ones, were absent. The fact that there was a slight decrease in the resistance to joint motion as the limp still became more apparent, was noted.

Still the limp increased, and so did the emotional symptoms. The limp became still more pronounced, but the other joint symptoms did not keep pace with the debility of the limb, while the hysterical were very prominent. This condition remained for a time, when the apparent urgency of some of the subjective symptoms led me to suspect that the emotional symptoms were secondary to a relapse or coincidental with it. I then asked Dr. E.C. Seguin to see the patient, and in a consultation, at which Dr. A. W. Catlin was present, it was decided that the symptoms warranted protection to the joint. Accordingly, the splint was again applied, with relief to all the symptoms, the hysterical as well as those which were, as the result proved, real, for the formation of an abscess at the end of about six months proved the correctness of Dr. Seguin's opinion. The case has done well ever since.

The insidious progress of caries sicca of the articulations in the first stage, might easily suggest neuromimesis, or some other lesion of the nervous system, as it did in the case which Esmarch reports, and to which reference has already been made. In caries sicca of the hip-joint there exists, when the disease first manifests itself, a limp, which is so slight as to be scarcely noted,—very slight resistance to extreme joint movements, and an indefinite expression of pain; sometimes no oral expression of pain at all. After these symptoms have existed for a time, they may wholly disappear for several weeks. When they again return, they are a little worse, and generally follow some slight twist or injury to the joint. A total remission of the symptoms may occur several times. In the child these symptoms are generally referred to "growing pains," "habit," etc., and in the adult it has frequently been called "rheumatism," "sciatica," "hysteria," or even in one case "malaria." I could relate many cases of this character did space permit. I can only refer in these general terms to this, the serious side of the question, for the error of diagnosing a joint lesion, when a neuromimesis exists, is as nothing compared with the evil consequences, the deformity, and death, which have resulted from mistaking a progressive and serious hip-joint lesion for "sciatica" or "rheumatism."

I have seen a large number of cases of simulated diseases of the spine, from the histories of which I select the following as affording the most instruction.

Case 9.—Miss ———, aged 14 years, residence, New York State, was seen in consultation September 28, 1879.

The history shows that the patient has always been a strong child. She is one of six children, three of whom are now living, one having died in early infancy, one of diphtheria, and a third of convulsions following measles. Two of the survivors have had convulsions. The patient has had rheumatic pains at various times in shoulders, hips and knees, which were, however, of but a few hours' duration.

The hereditary history shows phthisis on mother's side, one case of Pott's disease in a very remote branch of the family, and on the father's side, gout.

There was no trouble with the patient's back until five years ago, about which time, riding down hill in a little wagon, she fell therefrom, striking the lower part of her spine upon a stone. She was able to walk, however, and went to her room. A "black and blue" spot appeared at about the last lumbar vertebra; she was rubbed with liniments, kept quiet for a few days, recovered perfectly and remained entirely well for three years. Two years ago, when about twelve years of age, she began to ride on horseback, and became particularly active and venturesome. One day after a long ride she complained of her back, and the family physician was summoned. He thought "the muscles were strained." Iodine was applied, and rest prescribed in the recumbent position for a few days. Again recovery was complete, and the young girl was as active as before, except that the horseback rides were discontinued for about three months, when her father bought for her use a Shetland pony. He "bolted" one day and threw the girl over his head, again injuring the spine, which was treated as before with iodine and rest. But on this occasion other symptoms supervened. Practice at the piano tired her: "her back would ache between the shoulders." Practice was dropped. She became nervous and irritable; was very tired after slight exertion; desired to support her head when sitting down, and the spine became tender at various points. In this state of affairs the mother's attention was called to a case of Pott's disease in a distant relative. She became very anxious about her daughter, who became very anxious about herself. The tender spots along the spine became more tender, the spinal ache was more marked. The family physician admitted he was in doubt as to the condition of the spine, and the case was sent to a prominent surgeon of this city for advice. A diagnosis of Pott's disease at the last lumbar vertebra was made; the patient was placed on her back for three weeks, leeches and ice were applied to the spine, and she was then encased in a plaster jacket, after suspension, the operation being repeated five or six times in fifteen months, each time with relief. After the removal of the last jacket, however, the symptoms became worse, and the patient, naturally enough, objected to its renewal during the Summer. Her mother, in the meantime, had kept the patient very quiet, waiting for the cooler weather. When the patient applied to me for advice, the mother remarked that her daughter's symptoms were worse than ever before, and a fear existed that a relapse had occurred.

Examination.—Several hyperæsthetic spots over the spine, chiefly in the region of the 12th dorsal and 1st lumbar vertebræ; the pain, however, when present, was lower down, near the sacrum, and it had always been of a superficial character, the friction of her underclothing at the tender points hurting her more than the surgeon did by pressure when examining the parts. There was no pain or history of pain in the abdomen, thighs or pelvis. Careful examination showed normal flexibility of the spine in all directions, and there was no muscular resistance when the psoas and iliacus were put upon the stretch, with patient in the prone position and pelvis firmly held.

The spinal deformity showed itself principally in a marked excurvation, a slight drooping of the right shoulder and some lateral deviation. Lower extremities of equal length. All traces of deformity disappeared in the prone position.

Patient very evidently an emotional girl of active mind and of tolerably full habit. There is no evidence about her —in gait, attitude, or expression—indicative of Pott's disease.

A diagnosis of neuromimesis was made, and the patient was told, much to her surprise, that no apparatus was required. She was instructed to resume her equestrian exercise, and to do, in short, the same as other girls of her age in such matters. A letter recently received states that my diagnosis has been fully confirmed.

It seems scarcely necessary to comment on this case, and I would not make extended comment had not the error in diagnosis been committed by one whose opinion in such matters is generally accepted as authoritative.

1st. It has been stated again and again by various writers that superficial tenderness over the vertebræ is indicative of disease of the spine—a statement which other writers contradict.[1] The truth is that this localized tenderness does exist in many cases of true vertebral caries, but it is frequently developed by the surgeon at his examination, and has no real connection with the lesion. There is this difference between the tenderness of the subcutaneous tissues in Pott's disease and the hyperæsthesia of neuromimesis. In the former it is rarely made the subject of complaint by the patient, and, as before stated, is often developed by the surgeon at his examination—while in the latter condition it is one of the very first of which the patient speaks. The neuromimetic fastens on any abnormal sign—often of the most trivial character and exaggerates its importance; the victim of Pott's disease has enough to engage his attention without searching for superficial tender spots.

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[1] For example: C. B. Radcliffe, M. D., F. R. C. P., in Reynold's *System of Medicine* (vol. ii, p. 718, Article "Caries of the Vertebral Column,") describes among other symptoms of this disease "a feeling of undue heat, or even burning in the weak and painful and prominent part, which is not felt in other parts of the spine, when a sponge soaked in moderately hot water is passed down the spine—a state of tenderness on pressure or concussion, which is equally restricted to the same weak and painful and prominent part." On the other hand, Skey in the valuable work already quoted (p. 56), deals thus forcibly with these symptoms: "Of all the fallacies that cling to professional practice, of all the false doctrines which the pardonable ignorance of a former generation has entailed on modern surgery, none can surpass that which affects to detect a carious disease of the body of a vertebra by drawing the fingers down the spine." Again,

2d. It may be safely asserted that perfectly normal movements of the spine are incompatible with the existence of Pott's disease. Such flexibility existed in this case, and the incorrect diagnosis could only be made by giving undue value to some of the more prominent symptoms presenting. Even in those cases of neuromimesis where a certain degree of spinal rigidity exists, it will generally be found, as in the cases of joint disease I have described, that a persistent effort—not a forcible one—will overcome it. Nor must we mistake the acute and the sub-acute muscular affections for the reflex muscular spasm of chronic spondylitis. I have seen some cases, especially in the

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p. 55, he says: "Fifty or sixty years ago, a provincial surgeon of some note recommended the application of a hot sponge to the spine with a view to detect disease of the bodies of the vertebræ—there was some excuse for ignorance then—there is none now." My own experience leads me to wish that Skey's statements might find a place in every work on surgery. The frequency with which one hears the remark "There is tenderness over the vertebræ," proves that faith in this symptom as diagnostic of vertebral caries is still widespread. In true disease it has no significance whatever as a symptom of actual disease of the bone.

cervical region where a differential diagnosis was difficult, but in a great majority of neuromimetic spines you will find muscular lassitude rather than muscular rigidity: inability to hold the spine erect and a flexible excurvation rather than an alert muscular spasm which assists in protecting the diseased parts from concussion and aids in forming the compensatory, antero-posterior curves which are such a striking feature of caries of the vertebræ when deformity exists.

Case 10.—Miss M.A., æt. 14. Resides in New York State. The hereditary history of this patient is very suggestive: two of her grandparents—one on either side—had paralysis agitans: her maternal grandmother died of phthisis, a tendency to which exists in this branch of the family, two of her aunts dying therefrom, and her father's family are subject to chorea. Two of the patient's brothers have died of "brain disease," and a sister is "very nervous."

As a child the patient was "very quick and nervous," of active mind, and had no serious illness up to the age of eleven years. She seemed prone to assume responsibilities beyond her years, and sought the society of adults, and has always been a very affectionate and emotional girl. Three years prior to my examination of the case, she developed symptoms of a strongly emotional character which affected "principally the back." She had very severe pains in the dorsal region which prevented any movement of this part for several weeks. This was followed by several typical hysterical symptoms. While recovering from this condition, her little brother removed a chair upon which she was about to sit, and she received a severe concussion of the spine. The symptoms of trouble in the back now became very urgent: any movement of the vertebral column produced pain which, at times, seemed to encircle the body and frequently found its most marked expression in the abdomen and chest. The spine began to "project backward and twist sideways," and any jar or movement increased the pain greatly. The recumbent position was the only one she could tolerate, and she had kept it for many months prior to my examination.

Residing 400 miles from the city, she was brought here in a specially prepared compartment of a sleeping car, and was conveyed to her hotel on cushions. A diagnosis of Pott's disease had been made by the family physician, and strict injunctions had been given to avoid any shock or jar to the patient.

On examination I found a very marked excurvation of the spine which, however, immediately disappeared in the prone position, and a lateral deviation which yielded easily to manual pressure. The right scapula was lower and more prominent than the left. There were various points of tenderness over the spinous processes. Patient was unable to stand alone. Pain existed not only posteriorly, but anteriorly also—like the "gastralgia" of chronic spondylitis. There was no muscular rigidity, the vertebral column being normally flexible in all directions. There was no contraction of the psoas muscles, and the strictly involuntary symptoms which accompany true, chronic spondylitis were absent.

Good authorities tell us that pain on concussion of the spine, especially if accompanied by pain in the anterior part of the thorax, indicates Pott's disease, and it is certain that in typical cases these symptoms do exist. In this case they formed the prominent subjective symptoms, and if taken alone would be very apt to mislead. There was also a posterior curvature accompanied by lateral deviation, which latter not unfrequently occurs with the kyphos of Pott's disease. But the kyphosis, as well as the scoliosis, was easily removed, and instead of the ever present muscular rigidity of chronic spondylitis, was found a normal degree of flexibility of the spinal column.

A diagnosis of neuromimesis was not well received. The mother of the patient expressed herself as disinclined to accept it, so positive was she that disease existed. After some hesitation it was finally decided to follow my advice, which was simply to ignore the pain and the deformity and to accustom the patient to gradually progressive exercises. A tonic treatment was also advised. After three weeks the patient was able to walk, and after six weeks she went home suffering little or no pain. Progress after her return was gratifying. Recovery was complete without relapse.

The prominent symptoms of chronic spondylitis, before the appearance of the characteristic deformity, may be briefly described as follows:

1st. Rigidity of the vertebral column at the point of disease, this rigidity being, in a great measure, due to the persistent reflex muscular spasm which has already been described as occurring in chronic osteitis of the knee and hip.

2d. Pain, which, like the pain of hip- or knee-joint disease, may find expression orally, or give evidence of its existence by the apprehensive state already referred to. When the patient locates the pain, he generally describes it as occurring in the region to which the spinal nerves of the diseased region are distributed. The pain is aggravated by any sudden motion or unexpected jar. The same kind of involuntary symptoms, the nocturnal cry, etc., are found in many cases of chronic spondylitis in the prodromal stage. In some cases the disease progresses so insidiously that the actual deformity is the first symptom noted by careless observers.

3d. A characteristic attitude and gait, which are imparted, partly by the unremitting reflex spasm (which frequently obliterates the normal curves of the spine), and partly by the instinctive effort of the patient to avoid concussion or shock to the diseased surfaces.

I do not doubt that the same muscular atrophy occurs in chronic spondylitis that is found in chronic epiphysitis of the knee and hip. The difficulty of demonstrating it in certain muscles is apparent.

In neuromimesis of chronic spondylitis the pain is generally superficial, and is, almost always, located over or near the spinous processes; it is sometimes transient, and frequently changes its location from time to time. In Case 10 the pain appeared on the anterior surface of the body, but disappeared under the curative effect of exercise. In place of the reflex muscular spasm, which holds the vertebral column so rigidly in chronic spondylitis, and which prevents the reduction of the kyphos, there is generally found as before stated, a normal degree of mobility of the spinal column under properly directed manipulations. There is no nocturnal cry, and the facial expression of apprehension, which is generally marked in the sufferer from Pott's disease, is so far wanting that the expression of many neuromimetic patients is even merry.

The general conclusions reached regarding the knee-joint are as applicable to the spine as to the hip, excepting those, of course, which pertain exclusively to the knee as a distinct articulation.

If we recall the symptoms presented by the two cases of simulated chronic spondylitis which have just been related, it would appear that the danger of diagnosing true *lateral curvature of the spine* in similar cases would be great. Each of these patients presented a distinct lateral spinal curvature, accompanied by a malposition of the scapulæ, and these symptoms also occur in true scoliosis. Indeed, cases of nervous mimicry of true, rotary, lateral curvature are very frequent, and, inasmuch as the mimicry is often very close, we should, in suspicious cases, be very careful in our analysis of the symptoms presented. Fortunately, the error of calling a hysterical spine a true lateral curvature is not so likely to be followed by serious results, as is that of attributing to "habit" or "hysteria" the progressive curvature of true scoliosis, than which there is nothing in the whole range of orthopædic surgery more insidious in its onset, or, when it is fairly established, more difficult to arrest, even by means of the rejuvenated gallows, and the much lauded plaster-of-Paris bandage.

The almost constant association of emotional symptoms, of greater or less intensity, with the condition of true, rotary, lateral curvature, has led many to suppose that the former was, in some unexplained way, a factor in the production of the formidable conditions met with in the latter. My own experience leads me to say, while recognizing the fact that the majority of cases of true scoliosis are found in emotional girls, and that mental activity is its frequent attendant, that I am no more prepared to admit that this emotional state, or mental activity is the cause, or even a cause of true scoliosis than is the oft-noted precocity of strumous children a factor in the production of chronic joint disease. In either condition the emotional state, on the one hand, or the precocity on the other, is symptomatic of the general condition.

In true lateral curvature the dorsal curve presents to the left more frequently than is generally supposed, and in my own experience it occurs in males oftener than has been stated. Of 83 consecutive cases, occurring at the Orthopædic Dispensary and Hospital, 21, or 34 per cent presented a left dorsal curvature, and 18, or 27½ per cent were males. Adding to this the fact, which I deduce from the examination of several hundred patients, that in no two cases do we find exactly the same curves, and that it is exceptional, in a group of cases, to see the same number of vertebræ involved in the primary (dorsal) curvature, and we reach the conclusion that we must look for our primary muscular factor, in the production of true lateral curvature, to the, strictly speaking, intervertebral muscles, rather than to those which, taking their origin at some other part of the skeleton, have their insertion in the flexible vertebral column. I cannot stop here to enter at large into the field which this conclusion opens. My study of neuromimesis of lateral curvature has assisted largely in reaching this conclusion, which is secondary to other conclusions that are stated in my monograph on Pott's disease, already alluded to. I hope soon to be able to especially consider these and other interesting points in the etiology of lateral curvature, and regret that the time allotted me forbids my doing it on this occasion.

The fact that lateral curvature was very frequently accompanied by hysterical manifestation, is mentioned by Laycock. In his valuable treatise[1] he ascribes lateral curvature to "hysterical paralysis," and mentions the opinion of Stromeyer that the *serratus magnus* is involved in the production of the curvature, a view which has since been entertained by other writers. But there is this difference between the lateral curvature of hysterical origin and true scoliosis. The former partakes of the character of *functional weakness*, especially of those muscles which act upon the spinal column extrinsically, while the latter is due to a progressive muscular contraction, dependent upon causes yet to be pathologically ascertained, but which appear to primarily affect those muscles which act intrinsically. The hysterical form does not become true scoliosis, in my own experience, unless the specific, pathological cause be added, and we may, perhaps, infer that this specific cause is more apt to be developed in the hysterical diathesis, just as we may say that chronic joint disease is more apt to occur in the strumous diathesis. Whatever the pathological condition may be, that induces the peculiar condition known as rotary lateral curvature, we at least know that the muscular contraction is both a painless and a progressive one, and that it resembles in character that found in true torticollis, in congenital club-foot, and, in many instances, in infantile paralysis. The conditions found in true torticollis, especially resemble those which are apparent in true lateral curvature, and that this condition is one of *contracture* rather than simple contraction, is confirmed by Paget, who says, in speaking of neuromimesis of lateral curvature: "If these signs of distinction are not enough, ether or chloroform will help. You can straighten the mimic curvature when the muscles cannot act; you cannot so straighten a real curvature."[1]

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[1] "An Essay on Hysteria." By Thomas Laycock, Philadelphia, 1840.

Recognizing then the character and persistency of this contracture, it is always a matter of difficulty—if it is not an impossibility, in the absence of symptoms in the earliest stage—to determine just when the efficient cause of the progressive scoliosis commences to operate. When the spine is markedly curved, and rotation is apparent, the diagnosis is not difficult, and while the tendency of true scoliosis is to become very slowly worse, and to result in irremediable deformity, the hysterical curvature, if properly treated, sooner or later recovers, just as do the emotional contractions of the hip or knee.

The early stage of hysterical lateral curvature, and the first (apparent) stage of true scoliosis, however, present many features in common, and as before mentioned, the emotional element is almost uniformly present in each. This adds to the difficulty of diagnosis—and has led to many errors. For instance, a young girl applies for advice. The early history presents no remarkable features. She has always been well—but not rugged—has grown rapidly, and has become, of late, somewhat nervous and easily excited.

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[1] *Op. cit.*, page 229.

Mentally, the patient is quite active,—is fond of reading—and, maybe, is proud of her position in class. It may be also, that she is fond of out door sports, but on the one hand everything which the patient does is accomplished with a tireless sort of energy, or, on the other hand, she may go to the other extreme, and be very listless and languid. Her parents will tell you that lately the spine has become crooked. That one of the shoulder blades is quite prominent. The patient does not sit erect, and when she stands one leg is flexed, and "the hip" on the side opposite the flexed leg, is prominent. They fear that their child will become permanently deformed. An examination shows a curved spine, a prominent shoulder blade, a tilted pelvis with asymmetry of the trunk, especially between the free border of the ribs and the iliac crest on each side. There are tender spots over the vertebræ, hyperæsthetic areas that change from time to time, complaints of backache, sometimes headache. There may be also irregular or painful menstrual periods, etc. With a history like this before us, the question arises: Have we here a condition that demands mechanical support, or shall we simply remove all exciting causes and place the patient on her own resources? If it be a condition of true scoliosis, proper treatment cannot be too soon commenced, or too rigidly enforced. If it be a simple "hysterical spine" the course to be pursued is wholly different.

It is not unusual to find a history closely resembling the above developed in a true and unmistakable scoliosis. Generally, however, in this condition the symptoms are objective, rather than subjective. The spine is curved, the shoulder projects, but the patient has no knowledge of these signs herself. Her first intimation that her figure was not perfect, was derived from some closely observing friend, or the dressmaker. There may be no pain at all.

The disturbed muscular action, whatever be its cause, does its work painlessly, but none the less surely.

Rotation of the vertebræ is described by Barwell, Adams, and others as occurring very early in the history of true scoliosis. My own experience confirms this, and its value as a diagnostic sign in the first stage is very great. In my own experience also the first (apparent) stage of true scoliosis is always accompanied by a modification of the lateral flexibility of the vertebral column, in the dorsal region, while in the neuromimetic state, this modification does not exist. The loss of symmetry between the scapulæ in true scoliosis depends, mostly, upon the acquired position of the ribs, and in the mimic state, there is a simple debility of the scapular muscles, without any change in the ribs whatever.[1] In the simulated state there is generally an excurvated spine with a lateral deviation, while in true scoliosis the curvature is lateral wholly, with compensatory or double curves. And these facts enable us to mention the symptoms, which when found, render the diagnosis of lateral curvature certain, viz., rotation of the vertebræ, and marked resistance to lateral flexion on that side toward which the convexity of the dorsal curve looks.

Another point is this: Like the symptomatic lateral curvature found in chronic coxitis, or which arises from any cause which affects the transverse pelvic plane, the neuromimetic spine becomes straightened, and the scapulæ assume their normal relations when the patient is placed in the prone position. Absolute rest of the long, extrinsic muscles of the vertebral column is all that is necessary to restore the parts to their normal position. After very evident rotation and marked lateral resistance are met with, recumbency fails to wholly annul the curvature, though in removing the contributive cause of gravity, which acts vertically when the patient sits or stands, the position of the shoulder blades and the curvature itself becomes considerably modified.

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[1] Paget remarks "the vertebræ are little or not at all rotated, as they are in well-marked cases."—*Op. cit.*, p. 229.

But if, after examining a patient carefully, making accurate notes of your observations, you should still fail to reach a satisfactory conclusion, it may be deemed advisable to ask another examination at an early day. If during the interim any marked change in the symptoms occur, the evidence is strongly in favor of neuromimesis. There is "no alteration of increase or remission" in the muscular conditions, or the deformity, in true scoliosis. The shoulder blade does not change its position from day to day. The curvature and the rotation progress slowly, obeying the mandates of the incessant muscular contraction, but so slowly, that weeks may elapse without presenting any external change that the eye could appreciate. In the mimic state various and sometimes very marked changes occur in a few days. And if we find that these variations are not associated with the permanent features of the true scoliosis, which have already been mentioned, there is no doubt as to the existence of a neuromimetic state.

The treatment of hysterical lateral curvature will depend very greatly upon the extent of the debility of the extrinsic spinal muscles, and the consequent malposition of the spine and scapulæ. In many cases we can overcome the purely functional inertia of the spinal muscles, by suitable exercises, etc. But if the patient be a rapidly growing girl, either just approaching, or just past her menophania, more radical measures may be demanded. If the muscular lassitude be great, and the malposition a *constant* attendant of the standing or sitting position, it has been my habit to apply a very light and closely-fitting *elastic*, steel support. By its use we can succeed in antagonizing the dorso-lumbar excurvation, and thus secure the normal antero-posterior curves of the vertebral column. If the normal antero-posterior curves are successfully maintained, the lateral deviation as well as the drooping scapula seem to care for themselves.

This support consists of, 1st. Two light steel uprights, bent in the line of the normal curves of the spine, with the anterior dorso-lumbar curve slightly exaggerated. These uprights are then tempered; 2d. A pelvic band, reaching from trochanter to trochanter, to the center of which these uprights are riveted, as in the conventional spinal brace for Pott's disease; 3d. An anterior bandage or "apron," which secures these uprights and the pelvic band firmly against the body; 4th. The customary axillary pads, which pass from the upper termination of the uprights (about the second dorsal) to a crosspiece opposite the lower angles of the scapulæ. The resistance afforded by this simple support is sufficient to hold the vertebral column in the desired position, and yet the apparatus in no way interferes with natural or even graceful movements; and the muscles, though supported, are not deprived of exercise and use. This support also is worn a part of the day only, as a rule, suitable exercises, massage, faradism, etc., being used as circumstances indicate. Usually the support can be removed after a few weeks or months.

If space permitted, I might add cases illustrating neuromimesis of disease of the ankle, elbow, wrist and shoulder joints. And I have seen two or three mimic cases that might easily be called sacro-iliac disease, so closely did the symptoms coincide with those which have been recently described as being present in the first stage of this truly formidable lesion. It would be impossible, however, to cover the entire field of the nervous mimicry of joint diseases in one evening. The same rules, and the conclusions deduced from our study of the mimicry of knee, hip and spine diseases, are equally applicable to the differential diagnosis of the true and false lesions of other articulations.

Hysterical club-foot is not of very frequent occurrence, though a distorted ankle-joint may frequently accompany other contractions of an hysterical character at the knee and hip. Laycock states, "that local hysterical paralysis will give rise to a distortion of the foot,"[1] and Shaw[2] describes a case in which the "ankle was at this time turned round, so that she walked on the outside of her foot." Dr. W. J. Little, in his classical treatise on Club-Foot,[3] thus describes the position of the foot in a case of talipes varus acquisitus (hystericus) in a girl of 19: "its outer margin alone touching the ground, the sole presenting vertically backward, the tibialis anticus, tibialis posticus and gastrocnemii tendons being tense from contraction of their respective muscles, and toes firmly incurvated; she was unable to rest on the limb, the attempt being followed by an outward yielding of the tarsus, which brought the superior surface of the os cuboides to the ground. Locomotion could only be effected with crutches." Another case is also mentioned by the same writer: "Wherein contraction of the right anterior tibial muscle has been erroneously considered the sole cause of the distortion, and for the cure of which its tendon had been divided. I found that contraction of the posterior tibial muscle was the cause of the continuance of deformity."

Skey relates the following very interesting case:[4]

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[1] *Op. cit.*, page 130.

[2] *Further Observations on Distortion of the Spine.* By John Shaw, page 184, London, 1825.

[3] *Club-Foot and Analagous Distortions.* By W. J. Little, M. D., F. R. C. S., page 229. London, 1839.

[4] *Op. cit.*, page 104.

"In the year 1864 a young lady of 16 years of age was placed under my care under the following circumstances:

"For eight months prior to her visit to me, she had been suffering from inversion of her left foot, which was so twisted as to bring the point of the foot to the opposite ankle. . . . Her family consulted a surgeon of much experience in the treatment of distortions and of orthopædic notoriety. The case was considered as an example of an ordinary distortion, and the foot was placed in a very elaborately made foot splint, by the force of which it was made to approach a parallel relation with the other foot; but it was an approach only, for no mechanism could retain it in a perfect position, the toes yet in some degree pointing inward. Months elapsed, and the disease continued unchanged. A second orthopædic authority was then consulted, in conjunction with the first, and as no new light was thrown on the disease by the combined opinions of the two, the same principle of treatment was recommended to be continued, and the mechanism was somewhat more elaborated. . . . When the apparatus, which she had worn so long was removed on the occasion of her visit to me, her foot immediately resumed its twisted form. . . . The disease had appeared almost suddenly, in a person hitherto healthy. It could not be due to congenital deformity, and the limb gave no indication of disease or disorganization. There was neither pain, heat or swelling. In this case, also, there was no catamenial derangement.

"I removed the apparatus from the foot, bandaged the limb with a calico roller, ordered a full, nutritous diet, with bark and iron, and having explained the nature of the disease to a friend, sent the young lady home into the country, recommending her to rely on the kindly offices of nature—the greatest of all doctors, orthopædists not excepted. At the end of the month some progress had been made, but not a great deal. She still walked with some difficulty, but it was obvious that she was improving in health and vigor of system. At the expiration of six weeks she accompanied her family to a ball, her foot, as she entered the ball-room being not yet restored to its normal position. She was invited to dance and under the novel excitement she stood up, and, to the astonishment of her family, she danced the whole evening, having almost suddenly recovered the healthy muscular actions of the limb! She came to see me two days afterward. She walked perfectly well into my room and paced the floor backward and forward with delight. The actions of the limb were thoroughly restored, and all traces of the previous malady had disappeared."

Charles Bell also relates a case[1] in his work on "The Nervous System of the Human Body." Charcot mentions "a most interesting case," communicated to the Medical Society of Ghent, by Dr. R. Boddaert.[2] Adams dismisses the matter with the following remark: "A very severe and obstinate form (of talipes) is observable in young girls which is evidently connected with hysteria, and I need hardly say that in these cases the treatment must be directed against the general, rather than the local affection."[3] Brodhurst relates very briefly a case in which certain muscles were contracted, bringing "the outer edge of the anterior portion of the foot to the ground, the inner surface being raised, and the heel fully an inch from the ground,"[4]—but this condition was not permanent, and occurred during the catamenial periods only. Not to refer individually to all the authors who have written upon orthopædic surgery or club-foot, I will briefly state that the seeker after information upon hysterical deformities will be greatly disappointed, especially when consulting the most popular American authorities. Many writers on diseases of the nervous system also practically ignore this subject—or merely mention the existence of such conditions, and several works which I have consulted do not even refer to hysterical club-foot. As the nervous mimicry of club foot is not always easy of diagnosis, and as the treatment required is different from that which is applicable to the ordinary forms of talipes, the errors likely to ensue make a brief study of these cases profitable.

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[1] Referred to by Charcot.

[2] *Annales de la Société de Médecine de Gand*, 1859, p. 93.

[3] *Club-Foot. Its Causes, Pathology and Treatment*, by William Adams, F. R. C. S. 2d ed. London, 1873.

[4] Brodhurst on *Club-Foot.* London, 1856.

I have seen four cases of hysterical club-foot. In one of these cases there was contraction also at the knee and hip. In one (Case 12), the distortion came on after emotional excitement, and other symptoms which will be described. The third occurred in my private practice and was placed, at my request, in St. Luke's Hospital, where it was attended conjointly by my friend and colleague, Dr. G. G. Wheelock and myself. This patient, a precocious and emotional girl of 11, presented many peculiar symptoms, and was discharged unimproved. She afterward recovered at home, I am informed, under severe measures instituted by her father. The following case may, however, be called a typical one, and as ample opportunity was afforded for several examinations, I append the history in full.

Case 11.—In June, 1878, I was called to see Miss H——, aged 19, living on Long Island, at the suggestion of Dr. A. G. Thompson, of Islip. The patient was incapacitated by a club-foot—which was almost daily becoming more troublesome.

The history developed was as follows: In a hereditary sense there was nothing of special importance, except that a paternal aunt had "died from some lingering nervous disease." The parents of the patient were living and were in good health.

The patient had rheumatism when she was three years old, and for the three or four years succeeding this attack suffered from recurrence of the same disease. Up to the age of fourteen, when menstruation appeared, she had been otherwise healthy. At the age of fourteen a severe attack of inflammatory rheumatism occurred. In 1877, my friend, Dr. Charles W. Packard examined the patient and found her to be suffering from organic heart disease, a condition (aortic regurgitant), which was evident at the time I examined the case. The patient had never been especially emotional, and, to outward appearance, was in good health.

On the evening of February 22, 1878, she was seized with difficult breathing, pain in the region of the heart, and various, irregular, convulsive movements. There was, in the course of the next few days a repetition of these attacks, and they finally culminated in typical hysterical convulsions, with opisthotonos, etc. Various remedies were used, and after many fruitless efforts it was found that they could be best controlled by full doses of morphine. The effect of these attacks was to induce great debility. The patient could walk very well, but there was no deformity of the foot. Soon after, with no other manifestations of importance, the urine became suppressed, and for 72 hours there was, apparently, no urine secreted at all. When the bladder was emptied, there was found to be only a comparatively small quantity. Morphine accomplished more than any other remedy in temporarily restoring the secretion of urine, as in the case of hysterical anuria recently reported by Drs. McBride and Mann.[1] After this, on several occasions, hysterical symptoms manifested themselves, until, on April 21, 1878, the patient began to walk upon the outside of the right foot. There was no pain in the limb anywhere, except that occasioned by undue pressure over the cuboid bone.

After the appearance of the deformity the patient became greatly disinclined to take exercise of any kind, partly on account of the pain produced by walking, and partly on account of an avowed indifference. Her principal occupation had been embroidery or reading. At the time of examination she walked with a very awkward gait, limping very much, and the knee seemed to possess very little strength. The position of the foot was that of uncomplicated varus. The abductor pollicis pedis, the plantar muscles, the tibialis posticus and anticus as well as the gastrocnemius were very rigid, and the ankle-joint seemed actually anchylosed. I made various attempts to overcome the evident muscular contractions, but without avail. The position was not changed by manipulation during sleep. There was slightly reduced sensation below the knee, but no hyperæsthetic areas existed. There was no atrophy.

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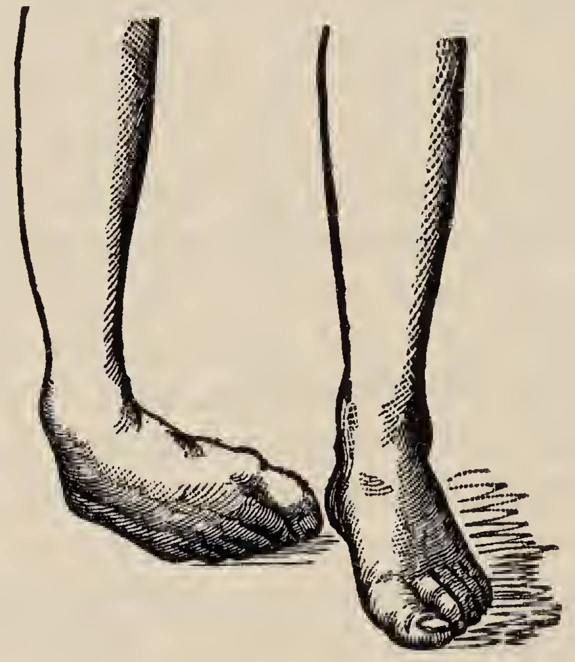
[1] "A case of Hysterical Anuria Cured by Restoring a Lacerated Cervix Uteri." *Archives of Medicine*, June, 1879.

A diagnosis of hysterical contracture was made. The warm weather was approaching, and it was deemed advisable to give the patient the benefit of a change of scene and air, with the hope that spontaneous recovery would ensue.

On September 12, 1878, the patient came to the city improved as to general health. Neither seashore nor mountain air had, however, affected the condition of the contracture. Before instituting any decided measures in the way of treatment, I thought it best to ask for a consultation, in view of the cardiac complication. Dr. E. C. Seguin examined the patient with me, and a course of treatment was decided upon, and the parents of the patient were informed as to the condition of the heart, and the possible effect of treatment, both upon the heart lesion and the deformity itself. After due deliberation, the father decided to make no effort in the way of treatment. The patient returned to her home, and has since remained tolerably well, though I am informed that of late, the opposite foot has shown a decided tendency to assume the same position as the one which was first deformed.

The position of the foot is shown in the accompanying engraving. As a matter of comparison, both feet are represented. It will be noted that, in addition to the varus position, there is a peculiar and extreme flexion of the toes.

In every case of hysterical talipes I have seen, this same peculiar flexion occurred, and a reference to Charcot's cases of hysterical contracture of the lower extremities, will demonstrate this same characteristic sign of hysterical affections of the foot. In true talipes this condition does not often exist, and its presence may be looked upon as an indication of the hysterical state.



The following case presents many interesting and instructive features, and illustrates the many phases "hysteria" may assume in boys of tender years, including even a neuromimesis of club-foot.

Case 12.—Willie M———, aged 10 years, residence Bridgeport, Connecticut. I saw the case first in consultation with Dr. D. H. Nash, September 29, 1877. Hereditary history good: no circumstance to note in early life.

While attending a military review in August, 1877, the patient fell, striking his back against the fluke of an anchor. The third lumbar was the point of injury and the evidences of the contusion caused thereby were evident for several days. While they were still present, about one week after the fall, he complained of weakness in his limbs, and, one night when ready for bed, he said he could not walk; he crawled up stairs on his hands and knees. The following morning he was much worse, was unable to stand alone and complained of great pain. The family physician was called in, who, after hearing the symptoms, asked "Have you hurt your back in any way?" Not an unnatural question under the circumstances, and the patient who had apparently forgotten the fall, recalled it and related the circumstances thereof to the doctor. The spine was examined and a tender spot discovered at the point of injury. Counter-irritation (croton oil) and rest were prescribed. The patient became worse: extreme pain in the back prevented his being moved in bed and soon he could not move his limbs. Then there was an apparent loss of sensation, for he professed utter indifference when pinched, or even pricked with a needle, especially in the right lower extremity; both limbs, especially the right, became cold. Such was the history given to me by Dr. Nash and the parents of the boy. I copy the remainder from my case-book.

Examination.—September 29, 1877. Patient in bed; countenance pale and apprehensive; complains very much at any attempt to change positon; severe pain located in back in region of third lunbar vertebra; pulse 95; auxiliary temperature, 98.4°. Is unable to move lower extremities, which show a reduced temperature as tested by Seguin's surface thermometer. Normal joint motion in lower extremities, except at the right hip-joint where a marked psoas resistance was met, such as is frequently found in the first stage of chronic spondylitis, in the region suspected; but this resistance yielded to a gentle and continuous force; rotation of thighs normal. At the expense of a good deal of pain, the vertebral column was proven to be normally flexible in all directions at the point of injury. The faradic reaction of the muscles of the left leg and thigh was normal, taking the biceps cubiti as the standard. On the right side, however, there was a very evident reduction, in the reaction of the quadriceps extensor cruris and the peronei. There was also a very considerable anæsthesia, especially of the right thigh and leg. A further examination of the vertebral column by palpation, demonstrated pain on slight pressure at the seventh cervical vertebra and at the point of injury, which still showed the peculiar eruption of the croton oil.

In the vicinity of the third lumbar there was marked hyperæsthesia; bladder and rectum normal, though the former had performed its function with some hesitancy. No cerebral symptoms; reflex movements as induced by titillation of the soles of the feet very considerably reduced on both sides. No apparent atrophy of either limb; limbs equal in length and circumference.

There was no history of fever, though early thermometrical observations had not been taken, and no other acute symptoms were noted than those above mentioned. The apparently excruciating pain was excited by any movement, especially by a sudden jar; it was with great difficulty that the patient was raised sufficiently to permit the use of a bed pan, and the bed linen though soiled, had not been changed for many days.

In reviewing the case in consultation, I declined to make a positive diagnosis with only one examination. Two conditions presented: a lesion of the spinal column (which had been strongly suspected) was eliminated by the normal flexibility of the spine at the suspected point and the absence of other symptoms which would positively indicate chronic spondylitis.

1st. An obscure lesion of the spinal cord, the early symptoms of which had been overlooked, leaving a slight sensor and motor paresis of the right leg. The fact that the external evidences of injury were located below the termination of the spinal cord was commented upon at the consultation.

2d. A neuromimesis. I inclined decidedly to the latter, and so expressed myself. I advised the continuance of the recumbent posture, tonics, and recommended a careful avoidance of any allusion to the patient's condition in his presence, and suggested that the counter-irritants be discontinued. The patient was to be lifted daily upon a blanket, and the bed linen changed. A second consultation was arranged for a few days later.

October 3d.—Upon entering the room, I proposed to move the bed so as to obtain a better light. To this the patient made urgent objection, and burst into tears. A promise that the bed should not be moved restored his equilibrium. Axillary temperature normal; pulse 85; no change in the faradic reaction of the muscles of either limb; anæsthesia still present; apparent psoas contraction which yielded to manipulation as before. The pain in the back was worse rather than better; appetite fair; bladder and rectum normal; but *the muscles of each lower extremity were very rigid*, and it required both time and tact to demonstrate that this rigidity could be overcome, but the patient's mind being diverted, this condition ceased suddenly, and free passive motion was permitted, but apparently at the expense of greatly aggravating the pain in the back. Adherent prepuce and masturbation had been eliminated from the case.

The irregular character of many of the symptoms, the exaggerated quality of those that did present with anything like constancy, the absence of definite indications of a cord lesion, and the certainty that there was no osseous lesion, determined the case, in my mind, to be hysterical. Feeling that I had the confidence both of the parents of the child and of the child himself, I deliberately lifted the boy from his bed, bore him across the room, and placed him in an easy chair by the window. Supporting the rigid limbs first with my hands, and then upon my knees, I called his attention to some passing object. The rigid muscles again relaxed.

After some assumed hesitation on my part, I permitted the patient to sit up for half an hour, watching him closely. The pain in the back disappeared, and when I left to return to town, he was sitting up in his bed, eating his supper with good relish. Instructions were given to make him leave his bed the following morning. He was to go out of doors every day, where he was to be allowed to do as he pleased. The following, quoted from a letter which I received from his father a short time after, will show the success of this plan of treatment: "Your patient sat up on Sunday (the day following your second examination), walked all about the house on Monday, was out of doors walking and riding on Tuesday, and every day since he has kept it up. He walks a little stiffly or weakly, but without a cane or any assistance." The improvement continued, and the boy soon walked naturally.

About three weeks later the father of the patient found it necessary to deny him some favor which he had asked. The patient sobbed convulsively, threw himself upon the floor and cried bitterly for upward of an hour. The father did not yield, and the child finally submitted and went to bed. The next morning a new symptom developed. The *left foot* assumed the position of talipes equino-varus. I was again summoned to examine the boy, whom I found playing ball, but walking very badly. Had it been the *right* foot that was affected, I would not have been surprised, for the peronei of the right leg had shown a diminished faradic reaction at both of my previous examinations. An examination, however, cleared the matter up: the peronei of the left leg showed normal contractility, the right alone giving diminished reaction. No other new symptom presented. Pain in the back had wholly disappeared, though the anæsthesia of the right leg still remained to a slight extent. The psoas contraction was gone. No attention was to be given to the "turning of the foot," and stimulated by a promise that he should have some money with which to buy Christmas presents if he overcame this trouble, it gradually disappeared, and has never returned.

The diminished faradic reaction, the anæsthesia, the unilateral psoas contraction and the greatly modified reflex action were the symptoms in this case that threw doubt upon the diagnosis. They are all compatible with the early hysterical state except the one first mentioned, and this condition remained after the boy's recovery.

Great care is necessary in determining the quality of the faradic reaction in suspected muscles, where the modification of the contractility is slight. My usual procedure was suggested by Dr. Seguin, and serves a good purpose. I use the five-post Kidder (tip) battery, and first find the normal reaction of the biceps cubiti. With this as a standard, I test the muscles of the sound limb, and the superficial muscles will generally respond to the same current. The cylinder of the battery is marked in fractions of an inch. I then test the suspected muscles with the same current, and increase its strength until the point of evident reaction is reached. A difference sufficient for diagnostic purposes can thus be demonstrated.

The treatment of hysterical contracture[1] is, as a rule,

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[1] I am indebted to my friend, Dr. Mary Putnam Jacobi, for the following extract from Duchenne's "*De l'électrisation localisée,*" 2d ed., p. 926. "The following is a remarkable example of a rapid cure of an hysterical contracture of the masseters, which had existed for two years." Obs. CCXIV (condensed). The contracture came on without any appreciable cause other than the hysterical state. Alimentation being difficult, an apparatus had been made which maintained the jaws slightly apart, and which was constantly worn. The contracture disappeared under chloroform, but it reappeared. It had disappeared spontaneously on one occasion, and had existed continuously for six months, when, after failure under the care of Drs. Campbell and Nélaton, it came under Duchenne's observation. The contracture disappeared entirely under the influence of "electro-cutaneous excitation"—two applications.

A case of hysterical contraction of the thumb, in a girl of ten, recently applied at the Orthopædic Dispensary. It had existed for several months. The patient wore, under my direction, a splint for about ten days. She was then transferred to Dr. Clovis Adam, Electro-Therapeutist to the very unsatisfactory, and the issue is sometimes doubtful. Many cases recover spontaneously, while others persist for years.

In one of Dr. Little's cases of hysterical club-foot, operative measures were completely successful. In other cases, after various remedial agents had failed, recovery has taken place at a time and under circumstances which involved an unexpected demand upon the volition of the patient, or under some form of emotional excitement. Purely mechanical treatment is apt to prove very unsatisfactory, and if used at all, preference should be given to cases of confirmed hysterical paralysis, where all other means have failed, or to the more exaggerated forms of hysterical spinal curvatures, where some simple, *elastic* support is indicated. And even in these instances, the use of apparatus should be made secondary to other measures. The application of mechanical force to overcome the deformities induced by hysterical contracture is positively contra-indicated. In the case of Miss H., Dr. Seguin proposed the use of atropia, hypodermically, in heroic doses, and the application of faradism, locally. The use of apparatus was not even discussed.

At his examination of this patient (Miss H.,) Dr. Seguin tested the electrical conditions of the muscles of the affected limb. The following is extracted from his notebook:—"External popliteal nerves react equally (both sides) to faradic current of weak power; muscles of anterior tibial and of peroneal regions react on both sides to same current, but right peronei contract less because of their stretched state. Galvanism (25 elements) to

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Dispensary. Three applications of a strong faradic current to the antagonistic muscles produced a complete recovery. And Dr. Adam also reports a case, in a woman of 25, of hysterical contraction of the hand and thumb, also completely relieved by similar measures.

external popliteal nerves gives normal and jerky contractions on both sides. Knee tendon reflex moderate and equal on both sides."

My own examinations of the muscles, made both before and after Dr. Seguin's tests, agreed with the above. I also found a normal response from the contracted tibialis posticus—the same current producing contractions on either side, the extent of the contraction being only less in the affected muscle, and this was due, I infer, to the fact that the muscle was shortened.

Charcot describes the prominent feature presented by this case as *permanent contracture*, and refers to it as being among "the most interesting peculiarities connected with the singular manifestation of hysteria."[1] He presents several cases which are fully detailed, and the conclusions reached are very valuable, and especially so to us in our study of the muscular conditions, as applied to deformities. This eminent writer says: "We have here a permanent contracture in the rigorous sense of the word. I have assured myself that it is in nowise modified during the profoundest sleep; in the day-time there are no alternations of increase or remission. The slumber alone which chloroform produces causes it to disappear if the intoxication produced be considerable. . . . The nutrition of the muscles has not sensibly suffered and the electrical contractility remains nearly normal."[2]

The diagnostic value of purely functional atrophy, of the effect of the anæsthesia induced by chloroform or ether, and the electrical test, in the conditions I have attempted to describe, are again made evident by Charcot's testimony. Indeed, in obscure cases, they form very valuable aids to diagnosis. It does not answer, as we have seen, to assume because a patient is hysterical, that all the symptoms partake of this character, however closely the obscure, but real, symptoms may resemble the nervous. And if, in a moderately emotional woman, for example, a condition of hysterical contracture should exist at the hip-joint, with other symptoms closely resembling hip disease, the difficulties of diagnosis can be very easily appreciated.

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[1] *On Diseases of the Nervous System.* New Sydenham Edition, 1877, page 283.

[2] *Op. cit.*, page 285.

Briefly then, we may summarize our observations upon the muscular conditions in joint disease, in the emotional *contractions* and in the hysterical *contractures*, as follows:

1st. In chronic osteitis of the articulations there exists a specific muscular atrophy, due to the lesion; an invariable muscular spasm—which is present night and day, and which, while not modified by the customary doses of chloral or opium, disappears completely under the anæsthesia induced by ether or chloroform. There is present also a marked reduction of the faradic contractility of the muscles thus affected.

2d. In the emotional contractions we find the atrophy of disuse only,—a variable muscular rigidity which disappears during natural sleep, or yields to opium or chloral—and a normal faradic contractility.

3d. In the hysterical contracture we see a "permanent" muscular rigidity, which like the muscular spasm of chronic osteitis, is wholly dissipated by the profound anæsthesia of ether,—but we find in connection with it, functional atrophy only, and a normal faradic reaction of the muscles.

4th. The test of anæsthesia induced by ether or chloroform, as applied to the differential diagnosis of hysterical contraction and chronic articular osteitis is not of value, *per se*, though some eminent authorities have stated otherwise. Ether or chloroform will remove the "permanent contracture" of the one, and suspend the reflex spasm of the other. The elements of absolute contracture, —such for example, as are met with in congenital talipes or torticollis, and intra- or extra-capsular changes (fibrous anchylosis, osteophytes, etc.,) being eliminated, we should bear in mind, in making our examination of suspected joints, under ether, Charcot's valuable deduction, viz., "that the existence of a spinal, organic lesion, of more or less gravity will be placed almost beyond a doubt, if under the influence of sleep induced by chloroform, rigidity of the members gives way slowly, or even persists to any marked extent."[1]

[1] *Op. cit.*, p. 297.

ON THE CARE OF CRIPPLED AND DEFORMED CHILDREN[1]

Read before the National Conference of Charities and Correction, New York, May 23. 1898.

So far as I can learn, the first effort to afford organized relief to the deformed and crippled among the poor of New York City was made in May, 1863, by the New York Society for the Relief of the Ruptured and Crippled. Under the leadership of its founder, Dr. James Knight, who gathered about him such men as James Lenox, Robert B. Minturn, Robert M. Hartley, John C. Green, Stewart Brown, A. R. Wetmore, Jonathan Sturges, James W. Beekman, John David Wolfe, Henry S. Terbell, and George W. Abbe, the work very rapidly took a prominent place among the great charities of our city. As I had the honor to be connected with the institution when it opened its doors for the reception of patients, in the very minor capacity of a medical student, and as I graduated in medicine under its auspices and filled the position of its first assistant surgeon, I can recall very distinctly the different stages of the development of this great work, and have watched, with much gratification, its progress and success.

Dr. Knight was a true philanthropist, a genuine friend of the crippled child, of more than ordinary versatility in adapting mechanical means to pathological ends in the treatment of the deforming diseases of childhood, and an almost unrecognized and neglected pioneer in the early work of what is now known as modern orthopædic surgery. In many respects he was a great man, and his self-sacrificing devotion to the work he founded has never met the recognition it deserves. I desire to place on record my humble appreciation of his unselfish efforts to relieve human suffering, and to emphasize the fact that it was owing to his energy and devotion that New York City first recognized the necessity of extending the hand of relief to the poor cripple.

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[1] *The New York Medical Journal*, for July 9, 1898.

Early in his work, and before the institution moved into its present ample hospital building, Dr. Knight recognized the importance of both mental and physical training as an important adjunct to the medical part of the work. He instituted a class in light gymnastics and calisthenics, and instruction, both religious and secular, was made part of the daily life of the patient. This work has been maintained since Dr. Knight's death.

The next important movement in the direction of the care of crippled and deformed children occurred in 1866, under the auspices of the New York Orthopædic Dispensary and Hospital. Its founders were Theodore Roosevelt and Howard Potter, who, with the assistance of Dr. John T. Metcalfe and Dr. Cornelius R. Agnew, secured the co-operation of Dr. Charles Fayette Taylor in forming this organization. Commencing simply as a dispensary, and with a board of trustees which comprised, among others, and including the founders, James Brown, William E. Dodge, James Boorman Johnston, Robert Lenox Kennedy, John L. Aspinwall, David Dows, Allen Campbell, Roswell D. Hitchcock, Robert Winthrop, and Charles Fayette Taylor, it soon became a factor in the charitable world, and rapidly assumed an enviable position both as regards its methods and its work.

The genius of Dr. Taylor in designing apparatus for the relief and cure of deformity made him a benefactor of his race, and his early retirement from active practice, through ill health, was a loss to the medical profession.

Mental training has always been a feature of the work in the wards of the Orthopædic Hospital. At present two teachers are employed: one in kindergarten work for the younger patients, the other in giving regular lessons to the older children.

As the writer stepped, so to speak, from a position in the Ruptured and Crippled Hospital to an almost similar one in the Orthopædic Hospital, and as his connection with the latter covers more than a quarter of a century, it is with peculiar satisfaction that he has watched the great growth of both of these institutions, which have done more than any other institutions in the country to afford relief to the crippled poor.

The next important step in this work of caring for deformed children occurred in 1872, when St. Luke's Hospital, attracted by the work of the Orthopædic Hospital, invited one of the surgeons of the latter institution to become an active member of its medical staff, creating for him the office of ''orthopædic surgeon.'' The orthopædic service of St. Luke's Hospital has since been one of its distinctive features, and it was the first large general hospital to recognize orthopædic surgery as a specialty.

Soon after this event there was a pretty general recognition of the necessities of the crippled and deformed on the part of many medical institutions. The New York Hospital, the Roosevelt Hospital, the Vanderbilt Clinic, the Postgraduate Hospital, the Polyclinic Hospital, St. Mary's Hospital, and other institutions opened their doors to crippled children, and organized either an outdoor or an indoor service, or both, for the treatment of the deformed.

In other large cities, and in some smaller ones, the growth of the movement to afford relief to the crippled and deformed has been very gratifying. In Boston, Dr. Buckminster Brown was placed in charge of a ward in the Samaritan Hospital, exclusively devoted to the relief of cripples, in 1861. This was two years before New York recognized Dr. Knight's effort, and the Children's Hospital of Boston is now a model in its orthopædic work. In Philadelphia the University of Pennsylvania has an elaborate and well-equipped orthopædic dispensary and hospital, founded largely by Dr. A. Sydney Roberts, and it also has other well-known institutions where the crippled and deformed are cared for. In Chicago, Baltimore, Rochester, Buffalo, St. Paul, Minneapolis, and elsewhere, the crippled and deformed are provided for, and all, or nearly all, this advance in the use of modern methods of caring for the deformed poor may be traced to New York, and to the personal influence of four great men—viz., Dr. James Knight, the philanthropist and organizer; Dr. Charles Fayette Taylor, the mechanical genius and enthusiastic leader; Dr. Lewis A. Sayre, the impressive teacher and eminent author, and Dr. Henry G. Davis, who revolutionized the treatment of joint and spinal diseases, and whose originality and genius made him the father of American orthopædic surgery.

It may, I think, be taken for granted, from a medical and surgical aspect, that the importance of caring for the crippled and deformed has been very generally recognized during the past quarter of a century the world over; though, strange as it may seem, the medical profession is still in many important medical centers, and especially abroad, unwilling to grant to orthopædic surgery the status it deserves. There still clings to the mechanical treatment of deformities in some minds the feeling that it is *infra dignitatem;* that to manipulate apparatus or to undertake to perform the necessary mechanical operations for treating deformity is, in some obscure way, outside of, strictly speaking, surgical lines. And, while this foolish prejudice is becoming modified, and especially so in New York, there are to-day in some sections of the country prominent surgeons who habitually refer patients with deformity to some commercial instrument maker, whose knowledge is limited to the shop in which he works, and whose education is far from equipping him to give the patient the relief which he has a right to demand. The day is coming when the instrument maker will occupy the same relation to the surgeon that the pharmacist does to the physician, when a "brace" for deformity will be a written "prescription" of the surgeon, and when the surgeon will know in detail what is needed for the relief or cure of deformity, and when he will rely upon his own experience and knowledge rather than upon the ignorant assistance and co-operation of a purely mercantile class.

Hence, in speaking of the care of crippled children, I desire to emphasize the statement that we need better educational facilities in our colleges for teaching orthopædic surgery—just as good, indeed, as are now given for other departments. Every medical college should have an orthopædic laboratory, just as there are laboratories for other necessary medical and surgical instruction. The student, under the direction of his teacher, should be taught, not that Smith's brace for club-foot or Jones's splint for hip disease is used in the treatment of these conditions, with perhaps a perfunctory demonstration of their application, but he should be educated in the principles underlying their utility, and should also be made proficient in their practical application. We all know how extensive and prolonged is the technical education demanded of those who, for example, are educated in the construction of a railroad bridge. The mechanical principles involved in the construction of a brace for spinal disease or club-foot are certainly not less important, and yet there is no, strictly speaking, technical education upon these points in any of our medical schools. The next progressive step in the surgical care of deformities will be to establish a mechanical laboratory where the practical use of apparatus in the treatment of deformities may be intelligently taught in all our medical colleges.

It is my intention, with the approval of the proper authorities, to establish such a laboratory in connection with the Cornell University Medical College.

Still, we all recognize how much has been accomplished since the pioneer institutions, especially the New York Orthopædic Dispensary and Hospital, have pointed out the way. The condition of the crippled and deformed has advanced in many important repects during the past thirty-five years. It rests with an organized body like that which I now have the honor to address, to study the problems and advance the interests of those who, if unrelieved, must become a burden upon the state, or, at least, be hopelessly relegated to the nonproducing class.

We must all recognize the fact that the average cripple needs much time for treatment, and that the period at which the most good can be done is at an early age, certainly before adolescence. There are some conditions, like knock-knee, bowleg, and club-foot, which may sometimes demand the direct (operative) surgical method, and which may not therefore require a long detention in a hospital; but many of the deforming diseases of childhood —for instance, Pott's disease of the spine (hunchback), hip disease, infantile paralysis, etc.—those which are the most disabling and the most chronic, are dependent upon causes which the knife can not always reach. They demand, many times—especially those dependent upon tuberculosis—years of active mechanical treatment, not, however, in bed, but in portative apparatus, which permits of gentle outdoor exercise. The question of education, or indeed of suitable mental occupation, during this period of enforced mechanical treatment and prolonged convalescence is a most important one. Some orthopædic hospitals have made systematic education a feature of their work, but much more remains to be done.

The medical and surgical treatment of the physical ills of the body should always be supplemented by a similar effort to educate the mind of the cripple, so that when he is cured he may meet his more fortunate fellows on an equal educational ground. But there are some deformities which are practically incurable, just as there are congenital deficiencies of the human frame which art may minimize, but science can never restore. This class has not received the attention it deserves in this country. As Dr. Augustus Thorndike, of Boston, in a most excellent essay on The Compensatory Education of Cripples, read before the New England Branch of the American Association for the Advancement of Physical Culture, and about to be published in *The Quarterly Journal of Physical Culture*, says:

> "There should be three sides to all education—mental, physical, and industrial—yet many of the homes and schools for cripples to-day confine their attention to only one side. There is no class of children more likely to be benefited by careful physical training than children left with a weak or shriveled leg or arm as the result of infantile paralysis or chronic joint disease."

In Boston, under the auspices of a board of trustees comprising Augustus Hemenway, Arthur A. Carey, E. Pierson Beebe, Miss Judith D. Beal, Joseph S. Bigelow, F. J. Cotting, E. H. Clement, Dr. Edward H. Bradford, Dr. Augustus Thorndike, and others, an Industrial School for Crippled and Deformed Children has been organized and has been in successful operation for three or more years. Dr. Bradford, in his report for the trustees, says:

> "It should be the work of an industrial institution for cripples to train the pupils for an occupation suitable to each individual case, and to furnish aid in finding work for those who may be capable of doing excellent work, but who, by their infirmity, are unable to solicit work."

It is not within the limits of this short sketch to detail all the admirable work done in this institution, whose patients, many of them, are still under careful orthopædic supervision, while being taught on the plan as outlined above. In many instances a congenital defect makes ordinary treatment useless. Among the inmates of this excellent institution is one, for example, who, having no arms, has learned to do typewriting with his toes.

I visited this school a few days ago with Dr. Thorndike, and found there much of interest and value. Forty children with various deformities were under careful mental and industrial training.

In Copenhagen there is a "Society for Cripples and Invalids" which has both hospital and dispensary departments. During 1895 there were treated by this society, paralytics, 177; those lacking one hand or a portion thereof, 117; other cripples, 119—total, 413. The following trades are taught: For men and boys, cabinet making, basket making, wood carving, turning in wood, shoemaking, and brush making; for women and girls, hand weaving, plain needlework, and dressmaking.

In Sweden and Norway there are four schools. Very much the same work is done in these schools as in the Copenhagen school. Special mechanical devices are used to enable paralytics to hold and work with various tools, and some pupils are even taught to work with their teeth, etc.

In various parts of Europe institutions of this kind exist, and their usefulness, especially in Italy, where deforming and disabling diseases are frequent, must be apparent; and among the many I might cite as having come under my own observation in this country, and as reported in Dr. Thorndike's exhaustive paper, is the Daisy Field Hospital and Home, located in Englewood, N. J., where children are taught to wash and iron their doll's clothes, to sew, to embroider, to make candy, canned fruit, etc., as well as to read and write; while at Philadelphia, at the Home of St. Giles the Cripple, basket making and carpentering are regularly taught.

This necessarily hasty and imperfect review shows America to be much behind Europe in the matter of industrial schools for cripples. It should not be so. In connection with every home and hospital where cripples are treated, the physical, mental, and industrial training should accompany the medical and surgical treatment whenever the combination is practicable; just as the moral and an adjusted religious training should enter into the school life of such societies.

Early in the history of the New York Orthopædic Dispensary and Hospital, in 1871, a system of outdoor visitation was inaugurated, it being recognized that much could be done if the interest of the patients was kept alive by periodical and friendly visits. This work was at first undertaken by a corps of lady visitors. Many patients not eligible for hospital care, and yet demanding minor but essential attention at home, rather than the skilled professional attendance provided at a hospital, were reached in this way, and much good was accomplished.

This system of visitation became such an important feature of the outdoor work that, when it outgrew the ability of the ladies to meet the demand, one and later on two surgeons have been employed to do this service, which supplements greatly that of the hospital; and some patients who might otherwise have relapsed after their discharge have been kept on the way to ultimate recovery.

Mr. C. Loring Brace, the secretary of the Children's Aid Society, writes me as follows regarding the Henrietta Industrial School at No. 224 West Sixty-third Street, New York City, under the direction of the Children's Aid Society:

"Our attention was called to the number of crippled children living in tenement houses who, because of the poverty and shiftlessness of their parents, were growing up almost entirely neglected. We determined to establish a class of these children in one of our schools as an experiment, and I think when you see it that you will agree that it is very successful. We purchased a wagonette and the children are brought to school in the morning and taken home in the afternoon by our teacher, receiving a lunch at the school. As we expected, we found children of all ages absolutely ignorant, and we have undertaken to teach them according to their needs, and it is possible that later on we may also teach them trades, so that they may be self-supporting. Different heights of tables, chairs, and all descriptions of reclining chairs, etc., are necessary in such a class. It is the duty of the teacher also to wash the children and rearrange their braces, etc., and advise with the parents regarding the children, and to be helpful in whatever way is possible. Especially it is the duty of the teacher to induce the parents to take the children to clinics and dispensaries for medical help.

"Our industrial schools are partly supported by public funds through the board of education and partly by contributions of charitable people, and the schools are under our oversight, but subject to the supervision of the superintendent of the board of education. This arrangement allows of much more individual care and much better acquaintance with the needs of the neglected little ones than if such a school were entirely under the charge of public authorities. The effort has been so successful that we propose to establish other classes like this one in other tenement districts as means permit."

I visited this school with Mr. Brace and found twenty-eight children with various deformities, under the treatment of various medical institutions, all of whom were receiving instruction, and all were happy in their work.

They belong to a class which can not, at present, be reached except by some organized body like the Children's Aid Society. Neglected at home, rejected by the public schools, incapacitated by physical deformity, and unable to care for themselves, this work, like the Boston home, supplies a very urgent need in the intelligent care of the crippled and deformed, and it must commend itself to all who are interested in this class of relief. As an aid to the outdoor departments of the various medical institutions designed to relieve the poor cripple, it is a necessity. I trust this school may be duplicated many times in different parts of the city and in different sections of the country at large.

Turning from this interesting and instructive side of the dispensary work, we may now consider a most important phase of hospital labor.

In a hospital devoted to the care of chronic deformities in childhood, the wards, after a time, become almost hopelessly encumbered with convalescent patients, who need only a minimum amount of attention, but who exclude the more acutely suffering from receiving the benefit of skilled professional care. In my own work I have often been confronted with a problem like this: A child with, say, hip disease or spinal disease is nearly well; perhaps the patient has been an inmate of the hospital for three or four years. He has no suitable home to go to, or perhaps, in effect, no home at all. If the patient is discharged, he is certain to lack even the slight attention which is necessary to keep the apparatus applied, and which is demanded in order to secure the best attainable result. If the patient be retained, a bed is occupied which is urgently demanded by an acutely suffering child with perhaps the same disease. What shall we do? On the one hand, we can not make a "home" of the hospital, and, on the other hand, if the patient is discharged, he is almost certain to relapse. Too frequently the patient is discharged to make room for another with the hope that a relapse, after all, may not occur. But this is all wrong.

The solution of this problem, it seems to me, is to have a country home, not too far removed from the city dispensary and hospital, under the care of a resident surgeon, where, with stated professional visits from the attending surgeon, and with specially trained nurses, the necessary medical supervision can be maintained, the mental, physical, and industrial training can be pursued, and the climatic treatment can be made to supplement the efforts of the surgeon.

I know that the question of such a country home has been seriously considered by both the Orthopædic and the Ruptured and Crippled Hospitals, and it is certain that if such an adjunct could be added to these institutions much permanent good could be accomplished.

And why should not the State aid in such an effort? A strictly dependent and even to-day a much-neglected class is being only half cared for by the excellent medical institutions established for its relief. The educational and charitable systems of the State should be adapted to meet the demands of this class of crippled and deformed as fully as are those for the deaf, the dumb, the blind, or the insane. And this is true from a medical standpoint also. A child with a curable deformity demanding prolonged treatment should be treated as well as taught until he is fully recovered, and not, when convalescence is fairly established and he is sure with proper care to recover, be sent out of the hospital to relapse, after a few weeks or months, and to become ultimately a more than useless member of society, perhaps a permanent burden upon the State.

The problems involving the care of crippled and deformed children can only be touched upon in a paper of this brief character. It is a subject well worthy the attention of this conference, and I regret that the time at our disposal is so brief that it can not receive more consideration at your hands.

:o:

COMMENT by GEORGE BLAGDEN, A. B., Chairman of the Board of Managers of the New York State Orthopædic Hospital for Children and by HENRY W. HARDON, A.M., LL.B., a member of the Board

After receiving his preliminary training in the Hospital for the Ruptured and Crippled, 1863–1868, Dr. Shaffer in 1870 entered the service of the N. Y. Orthopædic Dispensary and Hospital of which he was chief executive officer and surgeon-in-chief until 1898 when he resigned. He then set to work on a plan he had in mind to establish a state hospital for the care and treatment of crippled and deformed children.

For Dr. Shaffer to conceive of a new way to assist crippled children was a call to a new service. In spite of the demands of a large and exacting practice he made the time to prepare a bill establishing a hospital for this purpose and to secure its passage by the legislature. This was not accomplished, however, until he had overcome obstacles which would have been formidable to any one less convinced than he that there was urgent need for such a hospital and that it was a proper enterprise for the State to undertake. Strange as it may seem there were persons connected with the management of the existing orthopædic hospitals of New York City who made strenuous efforts to defeat the project. The foresight of Dr. Shaffer was not shared by them. It is estimated that there are still at least 1,500 poor boys and girls in the State of New York needing orthopædic treatment who cannot obtain it.

Minnesota was probably the first state to appropriate public funds for the treatment of these cases. It and other states now have hospitals for this purpose, but New York, stimulated by Dr. Shaffer's zeal, has the distinction of establishing for the first time a fully equipped state hospital for orthopædic cases.

For ten years Dr. Shaffer served it without pay as superintendent and surgeon-in-chief. He devoted his time and skill to its patients without stint, and no inconsiderable moneys, his own and those of persons who understood his work for the crippled poor and were glad to support it. When he laid down his duties he did so knowing that his undertaking was on a secure foundation, that the hospital had achieved fame, not only in America, but in Europe and that it had served as a model for similar public undertakings.

There could be no better monument to a scientist, philanthropist and physician.

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